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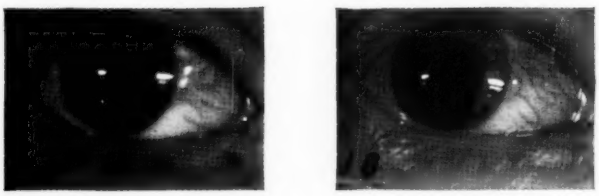

FIGS. 1A AND B [DAVIS ET AL]. LEIOMYOMA OF THE IRIS.

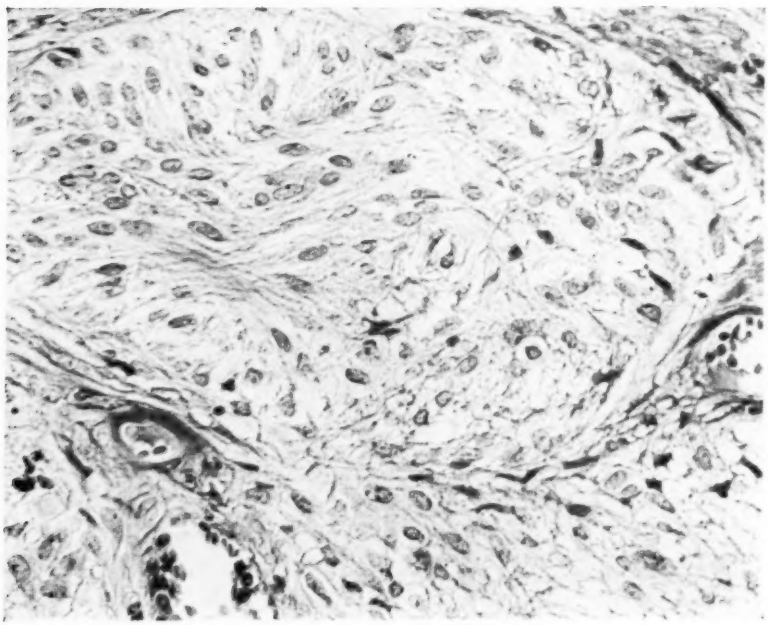

FIG. 2 [DAVIS ET AL]. LEIOMYOMA OF THE IRIS. MASSON STAIN ARMY MEDICAL MUSEUM ACCESSION NO. 87990.

AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 27 MAY, 1944 NUMBER 5

LEIOMYOMA OF THE IRIS*

A CASE REPORT

WILLIAM THORNWALL DAVIS, M.D., ERNEST SHEPPARD, M.D., AND WALTER J. ROMEJKO, M.D.
Washington, D.C.

Duke-Elder[1] referring to myomata states, "In the iris, they usually form sharply circumscribed tumors, appearing as yellowish-gray nodules without clinical symptoms, which grow slowly and are relatively benign, but if not completely excised, may recur (Verhoeff, 1936). Clinically, a myoma is quite indistinguishable from a malignant melanoma and for this reason the eye has usually been enucleated.

"While a myoma is impossible to diagnose clinically, its pathological recognition is by no means easy. The neoplasm is characterized by closely packed bundles of long, spindle-shaped cells with an eosinophilic cytoplasm and rod-shaped nuclei arranged in palisade-like rows (Fig. 2040): these are bound together with myoglial fibrils coursing along the cells and coalescing to form large fibers at their terminal processes (Fig. 2041). The picture resembles a spindle-celled malignant melanoma, the difference being that the cells of the latter are rarely distinctly spindle-shaped but terminate in and send off laterally, ill-defined processes anastomosing with those of neighboring cells to form a syncytium.

"It is only possible to distinguish between the two by differential staining (Mallory's phosphotungstic haematoxylin) and by the demonstration of the myoglial fibers by gold impregnation; and since these methods are comparatively recent, the cases reported by the older writers must be viewed with suspicion. The only three to be unquestionably demonstrated are those of Verhoeff (1923), Frost (1937), and Ellett (1939) (Fig. 2042). They seem to be associated with the sphincter muscle."

In Verhoeff's[2] case, the tumor was removed, apparently completely, on May 21, 1904. When the eye was dressed the next day it was found that a large portion of the tumor remained, giving the impression that none had been removed. Further attempts at removal with forceps and suction were unsuccessful. The eye was not enucleated until May 19, 1920, 16 years after the original operation. This was the first case in which the tumor was demonstrated by special staining. Of interest is its slight malignancy in that there was no material increase for 16 years. It arose from the surface of the iris by a constricted base and remained alive in spite of the fact that it was completely severed from the iris; its new blood vessels were derived from the wound. The original tumor did not invade the iris stroma nor involve the filtration angle. Histologically it showed no mitosis.

Frost's[3] case is the second "In which

*From the Department of Ophthalmology, George Washington University Medical School. Read in part before the Washington, District of Columbia, Ophthalmological Society, February 15, 1943.

the pathologic findings are sufficiently definite to justify this diagnosis." The eye had been examined periodically for six years, prior to the tumor's appearance in August, 1934, and was enucleated in March, 1935, because several consultants thought the tumor was a sarcoma.

Ellett's[4] case had had an irregular pupil for 5 years prior to the appearance of the tumor, which was of 15 years' duration. This eye also was enucleated because the tumor clinically resembled sarcoma.

Van Heuven[5] has reported a case of leiomyoblastoma of the iris, and Wassenaar[6] has reported a case of leiomyoma of the upper lid.

Collins and Mayou[7] state, "The points relied on in diagnosing these growths as myomata rather than spindle-celled sarcomata have been: the position in which they started, their slow rate of increase, the similarity in the shape and size of their cells to unstriated muscle fibers and the rod shaped appearance of their nuclei."

Reëxamination of Zentmayer's[9] case proved it to be a leiomyoma.

Case report

M. L., a woman, aged 27 years, was first examined on November 16, 1942, when she complained of a brown spot in the iris of the right eye which had been present as long as she could remember. She had injured the eye with a wire when a child, but of this there was no evidence. The history suggested a superficial scleral injury. The family history was irrelevant.

The ocular examination was essentially normal except for the tumor in the inferior temporal quadrant of the right iris, as shown in figure 1a. The physical examination by Dr. Lewis Ecker was normal. There was no evidence of primary tumor elsewhere. The blood Wassermann and Kahn tests were negative. There was 4 percent of eosinophiles, otherwise the hemogram was normal.

The tumor and adjacent iris tissue was removed on January 5, 1943 (fig. 1b). The postoperative course was uneventful. Pathologic reports were made by Col. J. E. Ash, Army Medical Museum, accession 87990, as follows:

"Gross: the specimen consists of a pale fibrous mass measuring 9 by 6 by 3 mm. There is an opaque elevated nodule 3 mm. in diameter on the anterior surface and brown pigment on the posterior surface.

"Microscopic: The tumor is composed of interlacing fascicles of non-pigmented, spindle-shaped cells with long fibrillar processes of smooth muscle character and oval, sometimes nucleolated nuclei. There is no evidence of intercellular collagen. The bundles of tumor cells are divided by an interlobular, richly vascular connective tissue stroma. The vessels are generally thin-walled but not of the sarcoma type. The tumor probably arises from iris muscle. Diagnosis: Leiomyoma, iris" (fig. 2).

Summary

1. Leiomyoma of the eye and its adnexa is rare. Cases have been reported involving the iris, ciliary body, and upper lid.

2. Of clinical importance is its slow growth and long duration, the absence of symptoms, the lack of any tendency to invade the surrounding structures or the filtration angle. With these characteristics it would seem advisable to consider excision before enucleation.

3. It is clinically impossible to differentiate leiomyoma from malignant tumors of the iris.

We wish to express our appreciation to Heleanor Campbell Wilder for kind cooperation in expediting the examination of the specimen and to Mr. Roy Reeves of the Army Medical Museum for preparation of the microscopic illustration.

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SUPPRESSION AMBLYOPIA*

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Although considerable attention has been paid to the subject of amblyopia, uncertainty remains with regard to its status. Views on its relation to squint have traversed the length of the pendulum's arc, as shown by Peter's[1] statement that "amblyopia in the squinting eye was formerly regarded as one of the causes of monocular squint but the lowered vision which is found is now interpreted as a symptom of squint and not as its cause." Conclusions drawn from a careful observation of amblyopia in patients with and without squint do not agree with either extreme. For this reason a statistical study was made of 200 consecutive amblyopic patients at the Barnes General Hospital.

The term "amblyopia" refers to a functional disorder characterized by a reduction of visual acuity which is not correctible to normal by optical means. Fuchs[2] has classified the conditions denoted by the term as follows: 1. Disturbances of sight seated in the eye itself—(a) congenital amblyopia, (b) amblyopia ex anopsia. 2. Disturbances of sight of central origin.

This classification has been used for many years but has several obvious flaws. First, there is considerable clinical evidence that so-called amblyopia ex anopsia is not seated within the eye itself but is of cerebral origin. The process of visual interpretation or "seeing" takes place in the brain, so any inhibition of the retinal image must be cerebral. Second, the term congenital amblyopia has been used to designate a condition which, according to Fuchs, "we assume to exist in those cases in which weak sight has existed for a long time, and in which all other causes for it can be excluded." In most of these cases the amblyopia would better be considered idiopathic. Third, Fuchs used the term amblyopia ex anopsia for amblyopia from nonuse resulting from such obstacles to vision as opacities in the cornea, lens, or pupil (pupillary membrane), or from disuse occurring in eyes affected with high refractive errors or squint since childhood. The amblyopia resulting from congenital opacities in the ocular media is not the same as that occurring in eyes

* From the Eye Clinic, Barnes General Hospital.

without pathology since it has been shown that the macula may not develop normally without the stimulus of the retinal image. In these cases the vision usually remains poor no matter how early the cataract is removed even after bilateral operations. If this type of amblyopia were acquired it would be expected that the vision would improve in one eye of patients operated on for bilateral congenital cataracts. In individuals with cataract acquired later in life the visual acuity usually is correctible to normal upon removal of the cataract even if the visual obstacle has been present for many years. The amblyopia due to congenital visual obstacles would more properly be termed *congenital amblyopia.*

A fourth possible objection to Fuchs's classification is the use of the term *ex anopsia,* meaning "due to not seeing" or "due to disuse." The question of its justification will be considered after a presentation of the results found in my series of amblyopic patients.

Abraham[3] has presented the following classification which is based on his view that the term ex anopsia is a misnomer and that suppression is the important factor. A. Congenital amblyopia. B. Acquired amblyopia: 1. Passive suppression (ametropic)—(a) bilateral; (b) unilateral. 2. Active suppression—(a) bilateral; (b) unilateral (squint).

Although I agree entirely with the use of the term suppression rather than ex anopsia there are certain objections to this classification. First, the same comment referred to in criticizing Fuchs's classification applies to the use of the term congenital amblyopia when idiopathic amblyopia is meant. Second, I do not believe bilateral-suppression amblyopia exists except in patients with hysterical amblyopia. The term suppression refers to a psychologic inhibition of the retinal image. When suppression becomes habitual, amblyopia is the result. Patients may have alternating suppression, as in patients with alternating strabismus, but amblyopia occurs only in one eye. Occasionally one sees a person with a high hyperopia which cannot be corrected beyond 20/40 in each eye; but this is a matter of lack of discrimination rather than suppression. It is frequent in young children to find a lower visual acuity than is commensurate with the refractive error. These same children later have normal visual acuity. At any rate, I do not believe subgrouping as to involvement of one or both eyes is justified. Third, Abraham considered passive suppressive amblyopia as due to lack of correction, or late correction of refractive error, and active suppression amblyopia as due to uncomfortable or confused binocular fixation. Whether such a subdivision is justifiable is another of the questions for which an answer is sought in the present study.

A classification which I believe more adequately divides the various types of amblyopia is simply:

(1) Suppression amblyopia (so-called amblyopia ex anopsia)
(2) Idiopathic amblyopia
(3) Congenital amblyopia (due to congenital visual obstacles)
(4) Discrimination amblyopia (bilateral)
(5) Hysterical amblyopia
(6) Amblyopia of toxic and neurologic disease

Only the first two groups concern us here.

Results

Of a total of 200 consecutive amblyopic patients 124 had no deviation even under cover, 63 were or had been convergent squinters, 8 were divergent squinters, and 5 had very fine corneal opacities which were in themselves not sufficient to account for the reduced visual acuity. Each group was subdivided into the following

categories, according to the refractive error found under homatropine cycloplegia:

(1) Anisometropia of at least 1.50 diopters of sphere, 1.50 diopters of cylinder, or both;

(2) High isohyperopia or isoastigmatism;

(3) Low refractive error or emmetropia.

The value chosen as the criterion of anisometropia was entirely arbitrary. It appeared more reasonable than the 0.5 diopters used by Worth and Lagleyze.[4] This, of course, makes a great deal of difference in the final statistical breakdown. Clinically an anisometropia of 0.75D. often causes discomfort, but suppression apparently does not occur unless the difference is 1.50 to 2.00 diopters.

I. Amblyopia without squint

The group of 124 nonsquinting amblyopic patients was divisible according to the following refractive conditions:

(1) Anisometropia 94 (75.8 percent)

(2) High isohyperopia or isoastigmatism 18 (14.5 percent)

(3) Low refractive error or emmetropia 12 (9.7 percent)

The anisometropia averaged 3.40 diopters of spherical difference in 69 cases and 4.02 diopters of anisoastigmatism in 25 cases. In those cases where both spherical and astigmatic anisometropia was present, only the higher of the two was considered. The remaining 30 patients had a difference in correction of less than 1.50 diopters of sphere, cylinder, or both. Of these 17 with high isohyperopia averaged 5.05 diopters of hyperopia in the amblyopic eye and 4.49 diopters in the nonamblyopic eye. The astigmatic correction in one case of isoastigmatism was 2.75 diopters each eye. The 12 patients with low refractive error averaged 0.99 diopter of spherical error in the

TABLE 1

Refractive correction in 98 cases of nonsquinting amblyopia

No.	Age	Amblyopic Eye		Nonamblyopic Eye (Corrected Vision 20/20 Each)
		Corrected Vision	Correction (Cycloplegic)	Correction (Cycloplegic)
1	22	20/200	+6.00+0.50×18	+1.25
2	32	20/200	+5.50	+4.00
3	25	C.F. 1′	−11.50+2.75×110	−1.50
4	22	20/200	+5.25+3.00×70	+1.25+1.25×75
5	23	20/100	+5.00+1.50×20	+1.00
6	21	20/100	+4.00+0.50×85	+2.50+1.00×85
7	21	20/200	+1.00+1.25×90	−0.50+0.25×90
8	24	20/100	+2.75	+0.75
9	21	20/200	+5.50+1.00×75	+4.50
10	24	9/200	+3.50	+0.50
11	22	8/200	+4.50	+1.00
12	40	20/200	+4.50+0.75×115	+1.25+0.50×90
13	26	20/200	−3.50+5.50×105	+1.50×60
14	21	10/200	+3.25+1.00×120	0
15	20	20/60	+3.50	+1.50
16	21	H.M.	+3.00	+0.75
17	22	C.F.	+5.00+3.25×75	+4.75+4.00×97
18	21	6/200	+6.00+1.00×120	+1.50
19	45	2/200	−2.50+8.00×125	+1.00+0.25×180
20	23	20/100	+0.75	+0.75
21	21	C.F.	−3.50+6.25×80	+1.00
22	22	20/200	+1.75+5.00×75	+1.00+0.25×90
23	27	20/200	+6.00	+5.50
24	34	20/200	+0.75	+0.50
25	22	C.F.	+6.00+2.25×100	+1.00
26	21	4/200	+5.00+1.25×90	+0.50
27	23	5/200	+3.75+1.25×90	+3.00+1.00×90
28	22	5/200	+7.50+0.50×90	+1.25
29	21	20/100	+1.25	+1.25
30	29	20/100	+4.75	+2.75+0.37×90
31	22	8/200	+4.50+3.25×60	+1.00+0.12×75
32	33	20/200	+6.00+1.00×180	+3.50+1.75×180
33	38	3/200	+1.00	+0.50+0.25×30
34	22	20/70	+3.25	+0.25×180
35	22	20/200	+4.75+1.25×165	+0.75
36	29	20/200	+8.00+0.37×165	+7.00
37	22	3/200	−3.50+0.50×45	+0.75+0.87×5
38	21	20/100	−3.00+6.00×85	−2.50+1.00×90
39	24	20/100	+5.75+1.00×110	+1.50
40	31	C.F.	+4.00+0.75×95	+1.75+0.37×70
41	20	20/40	+3.75+1.50×95	+1.00
42	24	20/100	+4.25+0.75×90	+3.50+0.50×80
43	25	20/400	+3.50+2.00×90	+0.75
44	20	20/40	+1.50+5.00×75	+0.75+0.25×90
45	19	20/400	+3.00+0.75×160	+0.25
46	—	20/100	+2.50+1.75×90	+0.50+1.00×75
47	30	C.F. 6″	+3.25+2.25×75	+0.25+0.37×75
48	23	20/40	+4.25+1.75×90	+1.50+1.25×95
49	25	20/200	+4.00+3.75×105	+3.25+0.87×80
50	21	6/400	+6.00+1.00×120	+0.50+0.37×90
51	32	20/200	+1.50+1.12×58	+0.75+0.37×105
52	22	L.P.	+1.50	+0.75+0.12×85
53	11	20/200	−3.50+2.25×15	−1.25+1.25×180
54	25	20/200	−3.50+8.00×105	+0.50+0.62×90
55	25	20/200	+5.00	+1.00+0.50×92
56	26	20/100	+3.75	+1.75
57	21	20/70	+3.00+1.25×90	+1.00+0.50×80
58	22	C.F.	+5.00+0.50×90	+0.75
59	36	L.P.	+6.50	+2.50+0.37×90
60	18	20/100	−1.50	+0.50+0.25×90
61	45	20/40	−2.00+3.75×135	+0.25+0.25×30
62	25	20/40	−2.50+3.50×140	−1.50+0.50×5
63	20	20/60	−2.50+3.50×105	+1.50×90
64	20	15/200	+3.00	+1.00+0.50×90
65	27	10/200	+4.50+0.25×180	+1.00
66	28	20/200	+3.00+3.25×75	+4.00+1.00×95
67	31	4/200	+4.50+2.50×55	+1.00
68	41	2/200	+7.00+1.00×180	+5.75+0.50×180
69	28	5/200	+6.25	+1.00+0.25×90
70	27	20/200	+3.50+0.50×10	+4.75
71	18	5/200	+6.50+1.50×130	+1.00
72	28	C.F.	+4.50+1.25×100	+2.50
73	19	3/200	+6.50+1.00×110	+4.25
74	24	20/200	+3.25+1.50×80	+0.75×85
75	46	20/30	−0.75+3.25×75	+1.25+0.37×75
76	25	20/70	+4.25	+1.50
77	26	20/70	+8.00+2.00×70	+7.25+2.00×100
78	22	20/50	+5.50+0.25×75	+3.00+0.75×125
79	25	4/200	−5.25+5.50×90	+0.75+0.75×100
80	23	5/400	+3.50+1.00×90	+3.75+0.50×115

TABLE 1 (*Continued*)

No.	Age	Amblyopic Eye		Nonamblyopic Eye (Corrected Vision 20/20 Each)
		Corrected Vision	Correction (Cycloplegic)	Correction (Cycloplegic)
81	32	H.M.	+1.00	+0.37×115
82	24	20/200	−1.25+1.00×40	+0.50+0.37×105
83	23	15/400	+6.00	+5.00
84	26	20/200	+2.75×75	+2.75×88
85	22	20/60	+1.12+2.75×85	+0.25+0.12×35
86	28	8/200	+6.50+1.00×90	+1.00
87	21	4/400	−8.00+1.00×95	−0.50+0.62×95
88	44	20/60	+3.00+0.62×110	+1.00+0.37×60
89	22	4/200	+4.75	+3.75
90	22	20/200	+0.75+5.50×80	+0.75+0.62×105
91	18	20/80	+1.25+4.00×100	+1.25+0.37×90
92	34	20/100	+0.75+0.25×90	+0.25+0.37×175
93	20	20/200	+5.25+0.50×100	+0.50+1.12×80
94	—	20/100	+2.00+0.62×95	+0.75+0.25×80
95	20	20/400	+3.00	+0.50
96	21	H.M.	+5.00+0.50×90	+3.00+1.00×80
97	24	H.M.	+6.00+1.50×85	+1.00+0.62×180
98	20	10/200	+5.50+0.25×15	+5.00
99	21	20/50	−2.75+5.00×85	−1.25+1.75×97
100	26	20/50	−2.50+3.50×90	0
101	35	20/200	+1.75+2.25×90	0
102	35	20/400	+5.50+3.50×103	+1.25+0.25×15
103	20	20/200	+2.00+0.75×75	+1.50+0.62×95
104	22	20/200	+0.75	+0.50
105	22	20/50	−2.50+3.50×15	−0.50+0.75×180
106	—	C.F.	+4.00+0.25×45	+1.50
107	19	3/200	+4.75+3.50×92	+1.50+0.50×90
108	24	3/200	+2.00+5.25×77	+0.75
109	27	3/200	−3.00+5.00×100	+0.50
110	23	C.F.	+0.75+5.50×80	+0.75+0.62×105
111	22	20/200	+4.00+1.75×75	+0.75
112	42	20/70	−8.00+1.00×90	0
113	24	20/200	−5.00+4.00×15	−1.25
114	26	3/200	+3.00+1.75×165	+0.75+0.87×5
115	20	20/400	+4.00×80	+0.50+1.00×105
116	22	20/40	+3.25	+1.00+0.50×85
117	18	20/400	+0.25×90	+0.25×90
118	23	20/60	+5.50+1.50×90	+1.50
119	20	20/200	+3.25+1.25×70	4.25
120	51	20/70	−0.75+0.75×85	−1.50+1.00×90
121	18	20/40	+6.00+1.50×50	+4.75+1.25×165
122	30	20/400	+5.75+1.25×90	+0.75+1.25×5
123	26	20/100	+3.00+2.50×95	+0.25+1.00×75
124	19	20/70	+5.50+2.25×10	+5.25+0.75×180

amblyopic eye and 0.65 diopter in the nonamblyopic eye. One of these patients had had the amblyopic eye patched for eight weeks during childhood because of lid disease. In this group suppression of the intermediate as well as of the central field of the squinting eye was present in those with less than 20/50 visual acuity (corrected) in the amblyopic eye. The tests were made with a stereoscope and Guibor cards.

II. Amblyopia with convergent squint

This group of 63 patients was affected only by comitant squints, some of which may have been precipitated by ocular pareses. They were separable into the following categories:

(1) Anisometropia 18 (28.6 percent)
(2) High isohyperopia or isoastigmatism 29 (46.0 percent)
(3) Low refractive error or emmetropia 16 (25.4 percent)

The anisometropia averaged 2.94 diopters of spherical difference in 16 cases and 4.12 diopters of anisoastigmatism in 2

TABLE 2

Refractive correction in 51 cases of amblyopia with convergent squint

No.	Age	Amblyopic Eye		Nonamblyopic Eye (Corrected Vision 20/20 Each)
		Corrected Vision	Correction (Cycloplegic)	Correction (Cycloplegic)
1	—	20/100	+3.50+0.50×95	+3.75+1.00×75
2	21	20/100	0	0
3	38	H.M.	+4.00	+4.00
4	28	H.M.	+2.25	+1.25
5	21	20/200	+1.50	+0.50+0.25×90
6	24	C.F.	−10.00	−0.50+1.00×90
7	26	20/200	+2.50+1.00×50	+2.00+0.50×110
8	21	C.F.	+1.25	+0.75
9	20	C.F.	+0.50	−0.25
10	22	20/100	+1.25+1.00×90	+1.25+1.00×90
11	21	10/200	+3.50	+0.75
12	21	6/200	+1.25	+0.75
13	21	3/200	+5.00+1.00×90	+0.75
14	25	20/100	+1.75	+0.50
15	21	20/100	+2.75+0.75×90	+1.00
16	22	20/200	+3.25+1.25×160	+1.25+0.87×50
17	27	20/200	+3.00+0.75×90	+2.50+0.50×180
18	20	20/200	+1.25	+0.50
19	—	20/100	+6.25+0.25×20	+6.00+0.25×180
20	21	20/50	+3.75+3.00×165	+4.00+0.75×75
21	—	20/60	+0.50	+0.75
22	26	20/200	+0.50+0.25×90	+0.75
23	36	H.M.	+7.75	+2.50+0.25×90
24	27	20/200	+3.50+0.50×90	+3.00+1.00×100
25	37	L.P.	+3.00	+2.00+0.25×20
26	38	20/300	+4.25+1.75×180	+4.25+1.00×10
27	21	20/200	+4.25+2.50×75	+3.75+2.50×102
28	22	10/200	+4.50+2.25×45	+3.50+1.75×120
29	26	4/400	+2.00+2.00×85	+1.25+1.12×85
30	26	H.M.	+1.75	+1.00
31	18	20/400	+3.25	+2.50+0.75×110
32	23	20/100	+2.50+0.75×75	+3.00+0.62×63
33	27	C.F.	+3.50+3.00×85	+3.50+2.00×95
34	28	20/200	+5.25	+2.75
35	27	10/200	+5.25+0.25×15	+6.00+0.25×165
36	26	H.M.	+0.50+2.75×90	−0.25+2.25×105
37	22	4/200	+0.50+4.00×180	+0.50
38	28	20/300	−1.50+1.25×135	+0.75
39	33	L.P.	+2.00	+1.75
40	21	20/60	+3.00	+1.00+0.25×135
41	—	20/80	+1.00+0.37×70	+1.00+0.62×105
42	24	10/400	+6.50+1.50×90	+4.50+0.25×90
43	27	20/200	+4.50+0.50×180	+4.25+0.75×180
44	5	10/200	+5.00	+4.50
45	24	20/200	+0.50	+0.50
46	49	20/400	+2.00+0.25×180	+2.00+0.25×180
47	25	20/200	+3.00+0.50×105	+1.25
48	26	20/60	+4.00	+5.00+0.50×180
49	22	20/100	+6.25+0.37×75	+6.75+0.37×120
50	19	C.F.	+2.00+0.25×105	+3.25+1.12×25
51	22	C.F.	+6.00+0.50×165	+3.25+1.37×25
52	19	L.P.	+0.50+0.50×90	+0.75
53	21	20/20	+3.00+0.50×175	+0.75+0.87×165
54	28	20/60	+2.75+0.50×90	+1.00+0.37×90
55	26	L.P.	+1.50+5.25×100	+4.75+1.00×90
56	25	20/200	+8.00	+4.75+0.25×150
57	20	20/100	+4.50+1.50×100	+4.75+0.50×90
58	24	H.M.	+5.50	+5.50+0.25×120
59	18	C.F.	+4.00	+0.75+0.50×95
60	27	20/400	+3.75+2.00×180	+3.25+0.62×50
61	25	20/100	+6.75+0.75×90	+5.50+0.75×115
62	31	20/200	+1.50	+0.25+0.75×90
63	24	20/70	+0.50	−0.25

cases. The remaining 45 patients had a difference in correction of less than 1.50 diopters of sphere, cylinder, or both. Of these, 28 had high isohyperopia averaging 3.83 diopters in the amblyopic eye and 3.67 diopters in the nonamblyopic eye. A single case of high isoastigmatism required a correction of 2.75 diopters of cylinder in the amblyopic eye and 2.25 diopters in the nonamblyopic eye. The remaining 16 patients with low refractive error or emmetropia required an average of 0.96 diopter of spherical correction for the amblyopic eye and 0.60 diopter for the nonamblyopic eye.

TABLE 3

REFRACTIVE CORRECTION IN 8 CASES OF AMBLYOPIA WITH DIVERGENT SQUINT

No.	Age	Amblyopic Eye		Nonamblyopic Eye (Corrected Vision 20/20 Each) Correction (Cycloplegic)
		Corrected Vision	Correction (Cycloplegic)	
1	23	20/70	−0.25	+0.25
2	22	20/200	+0.75+5.25×80	+1.00
3	21	H.M.	+7.00	+1.00
4	45	6/200	−5.50	+0.50
5	39	20/100	+3.75+2.25×30	+1.25+0.50×165
6	22	20/70	+0.25	−1.50
7	24	20/70	−1.50+1.75×105	−0.50+0.25×180
8	26	20/400	+1.50	+0.75

III. AMBLYOPIA WITH DIVERGENT SQUINT

This group consisted of only eight patients, separable into two groups: (1) Anisometropia 6 (75 percent); (2) Low refractive error 2 (25 percent).

The anisometropia averaged 3.81 diopters of spherical correction in four cases and 3.37 diopters of astigmatic correction in two cases. The two patients with low refractive error averaged 0.87 diopter of spherical correction for the amblyopic eye, and 0.50 diopter in the nonamblyopic eye.

IV. AMBLYOPIA WITH FINE CORNEAL OPACITIES

This group consisted of five patients, four nonsquinting and one with divergent squint. All were believed to have had good visual acuity in both eyes before the occurrence of corneal ulcer. They are separable as follows: (1) Anisometropia 4 (80 percent); (2) Low refractive error 1 (20 percent).

The anisometropia averaged 2.70 diopters of astigmatic difference in three cases and 2.50 diopters of spherical difference in one case. The single patient with low refractive error required 1.75 diopters of spherical hyperopic correction in the amblyopic eye and 1.00 diopter in the nonamblyopic eye.

The results may be summarized as follows:

	Anisometropia	High Isohyperopia or Isoastigmatism	Low Refractive Error
	no. percent	no. percent	no. percent
124 Nonsquinting amblyopia	94 (75.8)	18 (14.5)	12 (9.7)
63 Amblyopia with convergent squint	18 (28.6)	29 (46.0)	16 (25.4)
8 Amblyopia with divergent squint	6 (75)		2 (25)
5 Amblyopia with fine corneal opacities	4 (80)		1 (20)
Total	122 (61)	47 (23.5)	31 (15.5)

COMMENT

Of the series of 200 amblyopic patients, the condition of those (15.5 percent)

without significant refractive error may be considered as idiopathic. The discussion to follow will concern only the remaining group of 169 with significant refractive error.

If one considers the two large groups of the nonsquinting amblyopic and the amblyopic with convergent squint it is apparent that the chief difference is the predominance of anisometropia among the nonsquinters and of isometropic high hyperopia or astigmatism among the convergent squinters. The anisometropia was

TABLE 4

REFRACTIVE CORRECTION IN 5 CASES OF AMBLYOPIA WITH VERY FINE CORNEAL OPACITIES

No.	Amblyopic Eye Correction (Cycloplegic)	Nonamblyopic Eye Correction (Cycloplegic)
1	+0.25	−2.25+0.50×7
2	+0.50+3.50×115	+1.75
3	−0.25+2.75×60	+1.25+0.37×90
4	+1.75+1.12×45	+1.00+0.37×167
5(Diverg.)	−1.00+2.25×120	−0.50

more frequently spherical than astigmatic, in contrast to Abraham's findings.[3]

These findings suggest some causal relationships which must necessarily be largely theoretic. Our consideration depends in part on whether an eye must focus images sharply early in life to develop normally before the binocular reflexes develop. If this were true those infants whose eyes are highly astigmatic at birth would never be correctible to 20/20 with glasses later. If we assume that each visual element develops normally whether the image is sharp or blurred, then what Abraham calls passive suppression does not occur. With this assumption in mind let us consider anisometropia first. It must be agreed that aniseikonia exists in the uncorrected anisometropic. When the binocular reflexes begin to develop fusion is found to be difficult or uncomfortable. Psychologic inhibition of the disturbing macular image is resorted to. This process is known as suppression. As time goes on the suppression becomes more diffuse. If one imagines the visual field as an island shown in cross-section, representing the horizontal meridian of the retina (Roenne and Traquair), suppression involves the vision in a manner such as to level off the macular peak and eventually to depress the entire field. The periphery of the temporal field is least affected since it is not overlapped by the field of the other eye. When suppression becomes habitual amblyopia results. Frequently suppression may be reduced by occlusion of the nonsuppressing eye. However, this is not permanent unless the factors causing the suppression are removed.

An interesting type of meridional suppression in astigmatic anisometropia was described by Martin.[5] He found the visual acuity in the meridian of greatest diffusion to be one half or one third of that in the opposite meridian.

The process of suppression in anisometropia is often observable in patients following operation for monocular cataract. For a short while after operation the patient is conscious of the larger blurred image from the eye that has been operated on. In a relatively short time it is suppressed but may be called into consciousness if proper correction is placed before this eye. The same process is experienced by microscopists and watchmakers who learn to suppress the macular impression from one eye, at first voluntarily and later unconsciously.

In cases of high isohyperopia or high hyperopic isoastigmatism another process apparently goes on. In these patients there is a need for great accommodative effort in order to get clear vision. As the binocular reflexes develop accommodation and convergence become associated. The convergence resulting from the desire for clear binocular vision causes a

diplopia that is very confusing. To avoid this, suppression takes place in the more ametropic eye. With habitual suppression, amblyopia results. The fact that isometropic high hyperopia is most frequent among the convergent squinters fits in well with the accommodation-convergence theory just stated. In these cases convergence continues to be associated with accommodation even though amblyopia exists. This explains why the deviation in many cases of monocular convergent squint with amblyopia is reduced when convex lenses are worn. In other cases, if the hyperopia exceeds the ability of the accommodation to permit clear vision, no squint develops (Chavasse).[4] In my series of amblyopic patients the degree of hyperopia in those convergent squinters with isohyperopia was less (average of 3.83 diopters in the amblyopic eye and 3.67 diopters in the nonamblyopic eye) than in nonsquinters with isohyperopia (average of 5.05 diopters in the amblyopic eye and 4.49 diopters in the nonamblyopic eye).

In the cases of divergent squint, the majority were anisometropic. Suppression unquestionably preceded the amblyopia in these cases.

In the cases with fine corneal opacity the original ulcer had been present during early childhood. The suppression could have been produced either by the obstacle to vision at the early age or by the resulting change in refraction due to scarring, which usually produces anisometropia, particularly the astigmatic type.

During the period of binocular-reflex development, fusion may develop to various stages both in the anisometropic and in the high isohyperopic, but any sudden weakening factor such as shock or an acute infectious process may cause the attained level of fusion to be too difficult to sustain, so that recourse will be had to suppression. This accounts for the abrupt onset of many cases of strabismus.

In many instances of comitant squint, suppression precedes the deviation, whereas in others (probably including those with abrupt onset associated with illness or shock) suppression follows diplopia; so suppression cannot always be considered a result of the squint. In any patient with a paretic extraocular muscle the resulting diplopia leads to secondary suppression. Many cases of comitant squint are probably of paretic origin. Thus suppression and amblyopia may *precede or follow* the development of squint. This type of amblyopia *always follows suppression* and should be designated properly as *suppression amblyopia* and not as "ex anopsia." The process is always an active one rather than simply a passive loss of acuity from neglect, which the term "ex anopsia" implies. For the same reason Abraham's subdivisions into active and passive types do not appear to be justifiable.

Further evidence that the development of amblyopia is an active process is shown by the presence of a central scotoma which may be either relative or absolute. Uhthoff[6] found a scotoma in 55 percent of his cases whereas Heine observed it in 90 percent. Uhthoff was convinced that central scotoma is constant in marked amblyopia. Evans, Peter, and Travers have done extensive work in this field. Best[7] demonstrated that during microscopy an absolute central scotoma was present, without any involvement of the peripheral field. The improvement by occlusion and the transfer of the amblyopia from the poor eye to the better one by occlusion are additional convincing observations. The transfer of amblyopia apparently is possible only during the first few years of life.[8] That the age factor is important is shown also by the fact that

amblyopia rarely occurs in squinting children if the squint develops after the age of seven. It explains the relative infrequency of amblyopia in cases of divergent squint, most of which develop later.

It is obvious that amblyopia may or may not be associated with squint. Most nonparetic squints probably result from *suppression* plus an *additional factor,* either the accommodative stimulus to convergence, or simply the assumption of the dissociated position by the suppressed eye. Idiopathic amblyopia must be considered as due to suppression resulting from such factors as patching one eye in infancy or childhood and possibly hemorrhages due to birth traumata.

Conclusion

1. In disagreement with Peter's statement that amblyopia in squinting eyes is a symptom of squint and not its cause, suggestive evidence has been presented indicating that suppression and amblyopia precede the development of certain nonparetic squints and follows the development of other nonparetic squints and those of paretic origin.

2. Anisometropia is the predominating associated factor in nonsquinting amblyopic subjects, whereas high isohyperopia is the most frequent factor in cases of amblyopia with convergent squint.

3. Nonparetic squint probably requires suppression plus another factor for its development. This may be the accommodation-convergence relationship or simply the assumption of the dissociated position after suppression has been obtained in one eye. The age factor is obviously of considerable additional importance.

4. Suppression amblyopia is a more accurate term than amblyopia ex anopsia, implying, as it does, an active rather than a passive process.

5. The conclusions drawn may be of aid in adding links to the chain of evidence as to the cause of squint. Further clarification of the reason for normal muscle balance in a large number of amblyopic subjects, particularly a possible relationship to peripheral fusion, is necessary.

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EXTRADURAL DIPLOIC EPIDERMOIDS PRODUCING UNILATERAL EXOPHTHALMOS*

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Unilateral exophthalmos presents one of the most challenging diagnostic problems in ophthalmology. Among the unusual causes of this condition are the extradural diploic epidermoids. Such tumors are not to be confused with the so-called oil cysts or cholesteatoma of the orbit.[1] While both arise embryologically as a result of identical developmental defects, the point of origin of the diploic epidermoid results in characteristic bony changes which should aid greatly in the diagnosis of this condition.

In the neurosurgical literature the occasional reports describing these tumors have been collected by Bailey,[2] King,[3] and, most recently, by Rand and Reeves.[4] Since no such adequate description of this unusual condition can be found in the ophthalmologic literature; since, as a matter of fact, considerable confusion exists as to the actual nomenclature applied to the tumors under consideration, a summary of these papers would seem to add greatly to the worth of this report. The tumors in question have been described under the heading of "Pearly tumors," "Cholesteatoma vera," and "Epidermoids." This confusion is the result of the perpetuation of terms which entered the literature when the etiology and nature of the lesions were poorly understood. The situation is greatly clarified by sketching in historical perspective the development of our knowledge of the condition, and by bringing into focus controversial ideas as to etiology contemporary with the various stages in this development. In this sketch we have drawn freely from the aforementioned papers.

These tumors are epithelial in origin. They are thought to originate from epithelial-cell rests which become pinched off from the epithelial body covering during development. Such rests, remaining quiescent in early life, for some reason become activated at maturity. These primary epidermoids should not be confused with secondary epidermoids such as might result from the migration of epithelial cells through a marginally perforated ear drum, as is the result of blockage of a sinus after migration of such cells, or even as the result of the traumatic implantation of such cells within the diploic spaces. Macroscopically and microscopically there is no distinction between the two. The location of the growth in an area in which there could be no possible contact with mature epithelium is the distinguishing feature. The epidermoid differs from the dermoid in that its wall contains only squamous or basal epithelium. Such a tumor, lacking the sweat and sebaceous glands of the true dermoid, is potentially unable to develop into the so-called oil cyst of the orbit. It should present in all or in part the following characteristics: Theoretically the wall of the tumor should be composed of three layers. There should be an outer, relatively acellular, connective-tissue layer supporting a thinner, second, epithelial layer. The connective-tissue layer should consist of scattered fusiform and stellate fibroblasts embedded in a mass of intercellular fibers with few blood vessels. The second layer internal to that just described should consist of stratified squamous epi-

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thelium, the cell layers varying in thickness. The cells in the outer layers should be flattened, parallel to the surfaces, and should have no nuclei. Those composing the inner layers should be more cuboidal in shape and perpendicular in direction. Keratohyaline granules should be present in the cytoplasm of these cells, and for the most part the nuclei should be well preserved. Intracellular bridges may be seen. The inner third layer should consist of cornified epithelium. From this layer inward the epithelial débris accumulates as the cells are cast off. The lining membrane may be calcified so that the tumor is surrounded by an eggshell-like wall.

The first report of a tumor of such characteristics is credited to Cruveilhier (1829). In this report the growth was described as a "Tumeur perlée." By means of this term an attempt was made to describe the nodular and highly refractile surface of the tumor. In his report, Cruveilhier noted that two similar tumors had been seen by LePrestre and Dumeril, in 1807. In 1938, Johannes Mueller reported two tumors which fall within this group. Mueller was most impressed by the cholesterin crystals found in these cases and applied the name cholesteatoma. One of Mueller's two cases was the first reported case of what we now term extradural diploic epidermoid. Virchow, in 1855, reported four additional cases falling within this group. He objected to the term cholesteatoma, pointing out that cholesterin crystals were not a constant finding. In common with Cruveilhier, he elected the term "Perlgeschwülste" as being most descriptive. In the meanwhile, Von Remak, in 1854, first suggested that these tumors arose from embryonic epithelial-cell rests. These investigations, according to King, afforded the basis for the term *epidermoid* later used by Bostroem (1897). Cushing[5] (1922) observed that these tumors, quite rare as a group, occurred yet more infrequently when originating in the cranial diploe. The extreme rarity of the condition is further appreciated when it is realized that in Cushing's series of 2,500 verified intracranial tumors there were, according to Mahoney,[6] only 15 epidermoids—an incidence of 0.6 percent. The same author was able to find in the entire literature only 142 true epidermoids—of these only 23 were of the diploic type. Cushing reported one such case and collected from the literature six others. To the group he applied the term epidermoid cholesteatoma. More recently, Rand and Reeves have stated that: "From recent publications it is becoming increasingly evident that the terms 'dermoid' and 'epidermoid' are more frequently used in describing these neoplasms."

Diagnosis. Cushing, in 1922, stated that insofar as he knew the condition had never been diagnosed except at autopsy or operation. He also stated, however, that "They are capable of recognition roentgenologically, owing to the sharp bony defect and are capable also of complete surgical removal if approached by a flank rather than by direct attack in order that the epidermal membrane may be completely removed." Since the appearance of Cushing's paper, King, Olivecrona, and perhaps others, have on the basis of X-ray findings ventured preoperative diagnosis. The roentgenologic characteristics of the cranial defect produced by this condition are said to be positive and unmistakable. The outstanding and differentiating feature is the sharply defined, dense, white, scalloped margin found in no other condition. Any other eroding lesion, regardless of its nature, produces a defect the margins of which are poorly defined, hazy, or perhaps even fuzzy and soft. King further states that in a typical defect produced by the diploic type in

which the inner table is more involved than the outer, when viewed so that the greatest diameter of the defect is shown, the latter has a scalloped dense clear-cut margin, showing that this margin is more compact than the remainder of the skull. One or more bony hiatuses may be observed in the skull. These represent areas in which the outer table has been completely destroyed. If the roentgenogram is taken so that one views the defect "on edge," as though looking at a saucer edgewise, a dense line two or more millimeters wide is seen. This is due to the superimposition of the dense margins of the defect which brings the compact bony margins in alignment. The outer table may then be so thin as to be invisible in an underexposed film.

Other than the roentgenologic characteristics just described, there is nothing characteristic in the clinical findings. In 12 of the 14 cases of congenital epithelial tumor (dermoid or epidermoid) reported by Love and Kernohan[7] headache was the presenting symptom. In general, headache, mental disturbance, convulsions, palpable masses or swellings, disturbances of gait, vertigo, and disturbances of vision have been the presenting symptoms. Exophthalmos has been noted in only a few cases. One should be alert to the possibility of such a lesion if a patient presents himself with a painless swelling associated with obvious bone erosion which has increased very gradually in size without other signs and symptoms.

In consideration of the rarity of the tumor, especially when so situated as to produce exophthalmus we feel justified in reporting the following case.

Case report

E. S., a 24-year-old white woman, a textile worker, was admitted to Duke Hospital for determination of the cause of a gradually increasing loss of vision in the left eye of four years' duration. The past history was irrelevant insofar as any connection with the presenting signs and symptoms was concerned. At no time had there been evidence of inflammation in this area, nor any pain. There had been no suggestion of symptoms indicating increase in intracranial pressure.

Examination. The patient was a well-developed, obese, white woman, alert, coöperative, and in no distress. Except for the exophthalmos of the left eye, there was a normal configuration of the head and face (fig. 1).

Fig. 1 (Thornhill and Anderson). Exophthalmos due to cholesteatoma of the orbit.

The right eye and adnexa were normal. The marked exophthalmos of the left eye was especially impressive—measured with the Hertel exophthalmometer this amounted to 5 mm. (O.D. 15 mm.; O.S. 20 mm.). The veins of the lid were not dilated. No pulsations were felt. There was no bruit; nor was there pain on pressure or movement, or any limitation of movement. A soft, nontender mass, rubberlike in consistence was palpable under the supraorbital ridge. The ridge had lost its smooth, rounded contour and was sharply defined. On downward gaze there was a definite momentary distortion of the sclera in the upper portion which would persist for an instant and then round out. The pupils were regular, equal, and reacted to light and accommodation. Ophthalmoscopic examination of the right eye was essen-

tially normal. The optic disc of the left eye appeared paler than normal with blurred edges, probably due to the high astigmatism. The vessels were not unduly full nor tortuous. There were tension lines in the retina in the supratemporal region, radiating laterally from a center adjacent to the papilla. The peripheral fields showed no constriction. The right eye was emmetropic. The left eye required a −5.50D. cyl. ax. 180° for 20/20 acuity. No interesting nor pertinent information was obtained in the general physical examination of the body systems.

Accessory clinical findings: Hemoglobin 90 percent, white blood cells 11,360 with a normal differential, Wassermann test negative, basal metabolism rate −4, blood sugar 90 mg. per 100 c.c., serum cholesterol 208 mg. per 100 c.c.

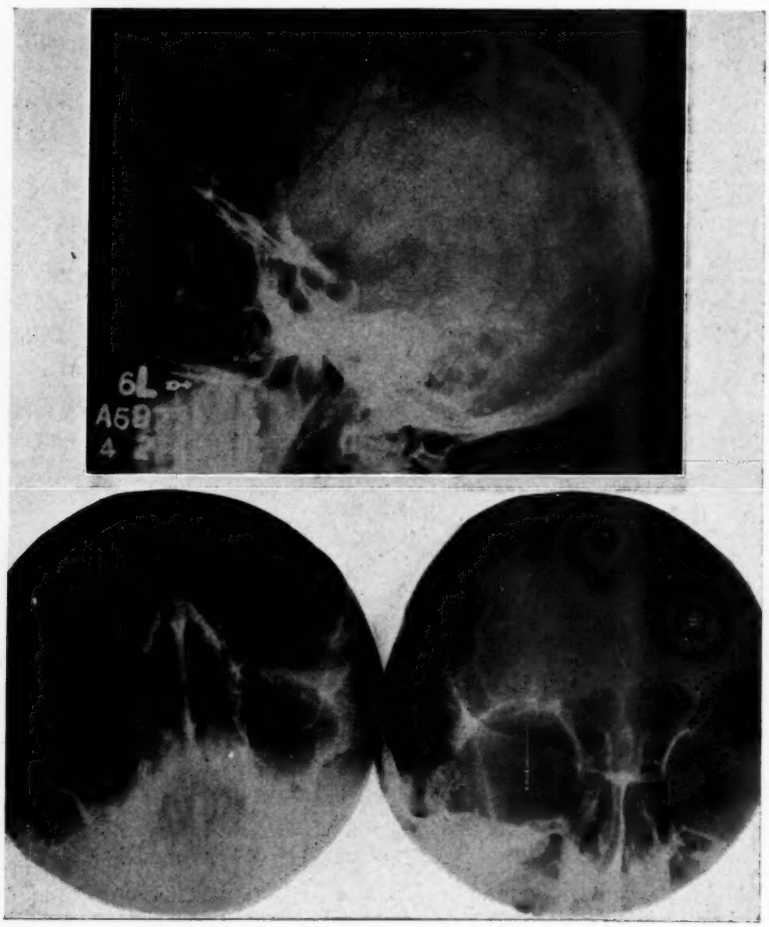

Fig. 2 (Thornhill and Anderson). X-ray studies in a case of cholesteatoma of the orbit.

X-ray (fig. 2) report was as follows: "Films of the orbit and optic foramina showed no erosion around the foramina. The upper wall of the left orbit has been destroyed, and an area of excavation extends upward measuring more than 2 cm. in diameter. There is no new bone formation, and the process must be differentiated between Schüller-Christian's

disease and a cholesteatoma."

Impression: Retro-orbital tumor (unclassified).

Operation. Under gas-oxygen-ether anesthesia the left orbit was explored through a supraorbital incision. As the periosteum was elevated an oval defect of bone was uncovered just above the supraorbital ridge and at the junction of its outer and middle thirds. The defect was approximately 1.5 by 2 cm. in size.

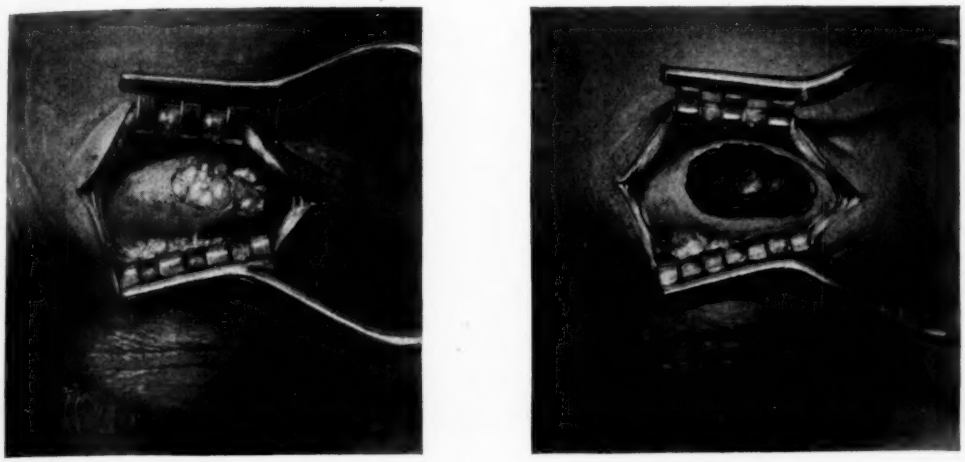

Fig. 3 (Thornhill and Anderson). The appearance of the mass exposed at operation.

Through it protruded a nonpulsating yellowish-white avascular mass. As the periosteum was further deflected three smaller perforations contiguous to that first exposed were uncovered. These were 2, 2, and 3 mm. in diameter (fig. 3). The bone lying between these dehiscences was of tissue-paper thickness. By removing these three intervening bridges the four defects were converted into one large opening through which an oval mass approximately 3 cm. presented. The surrounding capsule was quite friable and, in the manipulation necessary for removal of the tumor it ruptured. The contents thus exposed consisted of an outer layer of shell, 1.5 mm. in width, of white puttylike material laminated in concentric layers parallel to the friable capsule. In the more central portion the lamination became less conspicuous and finally indistinguishable from the central caseous core. These putty- and cheeselike contents were excavated with a spoon, after which the entire capsule was cleared away by meticulous piecemeal dissection. The inner table of the skull was found to have been completely eroded beneath this mass. The dura was intact but considerably depressed. The anterior superior wall of the orbit was also eroded and the contents depressed inferiorly. Careful search was made for any connection with any of the accessory nasal sinuses, but none could be demonstrated. The left frontal sinus was quite small and definitely medial to the area under discussion (fig. 2).

Immediately following the operation the eye receded to its normal position within the orbit.

The postoperative course was uneventful. Following operation the exophthalmometer readings were O.D. 15, O.S. 17. Vision was O.S. 20/300, with a −5.00D. cyl. ax. 180° it equalled 20/20.

One month later the exophthalmometer readings were 15 O.U. Vision was O.D. 20/20, O.S. 20/20 with +3.00 D.sph. ⊂⊃

−2.50D. cyl. ax. 180°. The fundus picture had not changed.

The *pathologic report* was as follows: "The pathologic specimen is material described as having been removed through the left orbit from the frontal region, and is said to have had the appearance at operation of a cholesteatoma. The material received includes approximately 14 gm. of tissue, which includes both irregular fragments of opaque, hyalinelike fibrous tissue and large irregular pieces of pinkish-gray, cheesy material. The irregular fragments break very easily on manipulation, compatible with the clinical description of the irregular hyaline strands which represent the capsular tissue; whereas the softer, grayish, cheesy material undoubtedly represents the evacuated contents. Grossly this tissue is compatible with the clinical diagnosis of cholesteatoma.

"The microscopic examination reveals for the most part two types of tissue. The first is represented by small irregular fragments of acellular, pale, eosin-staining, hyalinelike material. Nowhere in such fragments do we see anything which resembles connective-tissue cells nor anything which may be identified as epithelium. In some such fragments there is lamination with separation of adjacent keratinlike material. This tissue apparently corresponds to what was described at operation as the capsule. In addition there are also some fragments of tissue which may represent a part of this capsular tissue which has the appearance of elastic-tissue fibrils. There are also fragments of acellular, amorphous material in which no structure is evident. The latter tissue corresponds to the material so frequently seen within epithelium-lined cysts. However, nothing can be said in regard to its character inasmuch as material is not available for fat stains. There is no histologic evidence of cholesterol."

The microscopic picture of the tissue examined seems compatible with the clinical diagnosis of cholesteatoma. However, it must be pointed out that we are unable to identify the nature of the capsule and there is nothing to suggest either endothelium or epithelium.

Summary and Conclusions

Extradural diploic epidermoids producing unilateral exophthalmos are not usually considered among the possibilities in the differential diagnosis of the causes of this condition. None the less, the condition may exist in areas adjacent to the orbit and may be the cause of exophthalmos. The possibility that unilateral exophthalmos is due to such a lesion can probably be proved or eliminated by X-ray studies once the possibility is recognized by the typical scalloping of the edges with marginal increase in bone density demonstrated through proper angulation and exposure.

Complete removal and a cure was obtained in this case through the supraorbital approach. It is doubtful whether this particular type of tumor ever occurs so far back in the orbit (the tumor arises from diploic spaces which are not present in the walls of the orbit) as to justify the transfrontal approach recommended by neurosurgeons.

Box 3802.

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CONGENITAL CATARACT AND OTHER ANOMALIES FOLLOWING GERMAN MEASLES IN THE MOTHER

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In Australia in the summer of 1940 there was a severe epidemic of German measles. Overcrowding due to the influx of military recruits might have contributed to the rapid spread of the disease and its virulent nature. In the first part of 1941 an unusual number of cases of congenital cataract appeared in Sydney. Dr. N. McAlister Gregg published, in the Transactions of the Ophthalmological Society of Australia, a report on "Congenital cataract following German measles in the mother" and subsequently a nation-wide investigation was conducted by the National Health and Medical Research Council. The results of their findings are here reviewed and the author cites three similar cases seen in New York within the past few months.

GREGG'S FINDINGS

Gregg's report is based on 13 cases of his own, 7 of his colleagues' seen by him, with a total of 78 cases in all, including those reported by his colleagues to him for reviewing. The findings were as follows:

General. Most babies were small, ill-nourished, and difficult to feed.

Cataract. All of the babies had cataract from birth; all were bilateral cases except 16. The opacities were seen to fill the entire pupillary area when the pupil was undilated; but after dilatation two main types were observed. In the first, the contrast was marked between the larger, dense, pearly-white central area and the smaller, clearer, more peripheral zone. In the second, the density of the cataract was more uniform throughout. The process involved all but the outermost layers of the lens and was considered to have begun quite early in the life of the embryo.

Vision. The response to light was sluggish, but the babies appeared to follow readily any movement of the light stimulus.

Nystagmus. In young patients this was absent, but in older babies, or in patients whose treatment had been delayed, it was present. Movements were of a coarse, jerky, purposeless nature, rather than a true nystagmus. It was a searching movement, indicating an absence of development of fixation. It was present after treatment was delayed beyond the age of three months.

Variations. Three patients had corneal haze denser in the center than in the periphery. After two weeks the cornea cleared and the typical cataracts were seen. In one of these cases, the mother developed cataract during the pregnancy at the age of 27 years.

Monocular cases. There were 16, of which 10 presented microphthalmia.

Heart. Of Gregg's 13 cases all but one

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baby had a congenital heart condition. In the series of 78 cases, a congenital heart lesion was present in 44, while in many others the question was equivocal, evaded, or suspicious. Autopsy findings in three cases revealed a large patency of the ductus arteriosis, and the author understood that in autopsies performed elsewhere a similar condition was found.

Deaths. In the entire series 15 deaths were recorded. Several were from bronchopneumonia, and in three there was a sudden rise of temperature to 105° or even 106°F. followed by death within 24 hours.

Intolerance to atropine. In not a single instance has it been possible to continue the administration of atropine throughout treatment. Even after one or two instillations the patient exhibited considerable constitutional disturbance, with pyrexia, restlessness, irritability, and difficulty in feeding. In some cases the iris had an atrophic appearance. Full mydriasis could not usually be obtained.

Etiology. By a calculation from the date of the birth of the baby, it was estimated that the early period of pregnancy corresponded with the period of maximum intensity of the very widespread and severe epidemic in 1940 of the so-called German measles. Special attention, therefore, was paid to the history of the mothers during pregnancy, and in each new case it was noted that the mother had sufferred from German measles early in her pregnancy and, most frequently, in the first or second month. In some cases, at the time of the disease she had not realized that she was pregnant.

Incidence of German measles in series. In all but 10 of the 78 cases a history of German measles during pregnancy was obtained. In 2 of the 10 the report of measles was negative. In one there was a history of kidney trouble. In two the history was not asked for. In the remaining five the report is "no history of measles" or "not known." The majority of cases occurred in 1940 or early 1941. In Gregg's own 13 cases, the history of only 1 was negative. In this case the mother stated that she was kept so busy looking after 10 children that she could not recollect any details of her own health beyond the fact that she was ill at about the sixth week of pregnancy when one of the other children died suddenly from whooping cough. Even though ill, she was unable to go to bed during the last month before the baby was born, one month prematurely. In the great majority of cases the infection occurred either in the first or second month of pregnancy. In a few cases it was in the third month.

Nature of the epidemic in 1940. It was severe and accompanied by complications, and, in the majority of cases in this report, the maternal infection occurred in July or August of this year. Occurring concurrently with this epidemic were epidemics of sore throat emanating supposedly from military camps and spreading to the civilian population. The author questions whether or not the sore throats were streptococcal in origin and the rash diagnosed as "German measles" was a toxic erythema accompanying the streptococcal infection. The description of this rash by the physicians during the epidemic was pleomorphic. Out of 35 cases in which sufficient records are available, the affected baby was the first child in 26 instances, and in three others it was the second child. This notably high incidence in the children of primiparae indicates that the epidemic affected younger people.

Management. Babies should be operated on immediately to permit sufficient light stimulus to reach the retina so that fixation may be developed. If the stimulus is insufficient or delayed, nystagmus will result. The only contraindication to early operation is the general state of the baby's health.

Operation. The anterior chamber is

particularly shallow, and in many cases the very dense central portion of the lens has proved resistant to the needle. Sometimes it has separated off as a firm disc. In others, the whole lens has tended to move away from the point of the needle. In other cases the discission has been straightforward and easy. Absorption has been slower than that of the ordinary congenital cataract. No opportunity has so far been afforded to examine the fundi of any patient after absorption of the lens matter.

Prognosis. At the present state of our knowledge, we cannot state whether or not other ocular defects are present, or whether they will develop.

REPORT OF COMMITTEE OF NATIONAL HEALTH AND MEDICAL RESEARCH COUNCIL. Under the aegis of the National Health and Medical Research Council, a circular letter was sent by Drs. Swan, Tostevin, Moore, Mayo, and Black to all medical practitioners in Australia to obtain data about children born of mothers who had suffered from exanthemata during pregnancy. When possible, permission was requested to interview the mother and to examine the baby. In the metropolitan area, when a pregnant woman developed rubella or morbilli, it was asked that the investigators be notified so that an interview and examination could be carried out and the child subsequently born examined. If the patient lived in the country, it was requested that data be supplied after the birth of the baby.

Results of the investigation. The mothers were divided into the following groups: (a) those who suffered from rubella during pregnancy, 49 cases; (b) those who had no knowledge of any exanthemata during pregnancy, 4 cases; (c) those who contracted morbilli during pregnancy, 9 cases; and (d) those who suffered from mumps during pregnancy, 2 cases.

Summary. Of 61 infants examined in the course of this investigation, 36 were found to have congenital defects.

Of the 49 mothers who had rubella during pregnancy, 31 of the infants subsequently born exhibited congenital defects. The abnormalities included cataract, deaf-mutism, heart disease, microcephaly, and mental retardation. Eye defects were noted in 14 cases—13 infants had cataracts, of which 10 were bilateral and 3 were unilateral; 1 had buphthalmos. Seven had deaf-mutism; 17 had cardiac abnormalities; all had some degree of microcephaly. With two exceptions all of the 31 mothers with congenitally defective children had contracted rubella within the first three months of pregnancy. The investigators concluded that the disease of the mother was rubella and not a type of streptococcic throat, thought by Gregg to be a possible factor. They were unable, however, to explain why these new complications of what was supposedly rubella asserted themselves unless the virus had altered. The epidemic of German measles in 1940 in Australia was particularly severe, and it may be that this severity was caused by the war and conditions where large numbers of susceptible recruits were herded together in military camps. The disease spread rapidly and the causative agent may have reached a higher stage of virulence. Other investigators have shown that avian and other mammalian embryos show, in contrast to adult tissues, a particular susceptibility to infectious agents. The authors feel that it is logical to assume that the human embryo possesses the same susceptibility to infection that avian and other mammalian embryos do, and that the etiologic factor of German measles, after penetrating the chorionic barrier, is capable of producing severe lesions in the embryo, whereas the same infection in the adult tissues of the mother leads to only minor effects.

From this investigation it is claimed

that when a woman contracts rubella within the first two months of pregnancy, the chances of her giving birth to a congenitally defective child are about 100 percent, and if she contracts rubella in the third month, the chances are about 50 percent.

In a personal communication to the investigators, Gregg stated that all his patients were retarded in their mental growth if not mentally defective. For this and other reasons, Gregg thought that few if any of the children with both eyes affected were likely ever to develop into completely normal children. Other observations in the investigation included the occurrence of deaf-mutism, sometimes with heart disease, of heart disease without other apparent defects, of microcephaly, and of hypospadias.

The investigators concluded with the suggestion that the next step in dealing with this problem should be to isolate the causative agent of rubella with the object of preparing a protective vaccine against the disease. In the meantime, the effect of repeated doses of convalescent serum should be studied not only on mothers in the incubation period of the disease, but also as a prophylactic measure for any pregnant woman who has not previously suffered from rubella. At least this should be done to protect the mother during the first three months of pregnancy.

Cases recently observed by the author

In the past three months the author has had three patients who belong to the group described in the Australian reports.

Case 1. A. S., a girl born at full term without instruments, and weighing 5 pounds and 10 ounces, was examined on the third day because the parents noted a white reflex through the pupil of the right eye. Examination showed a dense, white cataract occupying the entire pupillary area and preventing any fundus reflex. The pupil was small (from 2 to 3 mm.), and reacted slightly to light. The anterior chamber was shallow. The cornea was clear and the iris appeared normal. The intraocular pressure to palpation was normal. The entire eye seemed somewhat smaller than the fellow eye, and this explained the fact that the right palpebral aperture was narrower than the left. On the fourth day the baby had an attack of cyanosis and was given oxygen. This cyanosis was thought to be due to a congenital heart lesion. There was a murmur over the entire precordium, and X-ray films showed an enlarged heart. No subsequent attacks of cyanosis have occurred. There has been no feeding problem. No microcephaly was present. When the baby was six weeks old, there was a severe conjunctivitis of the right eye causing redness, swelling of the lids, and a mucopurulent discharge. This cleared up in a few days but was followed by high temperature, and meningococci were identified in the blood stream and in four joints. The patient survived this and in one month had an infectious diarrhea which has now cleared up. The baby is now three months old and weighs 7 pounds and 11 ounces. The mother had German measles in the third week of pregnancy. (I am indebted to Dr. Rich Siegel, the child's pediatrician, for data on the general condition.)

Case 2. G. D., a girl, born 12 days early without instruments, and weighing 5 pounds and 15 ounces, had, since birth, a dense white cataract in each eye. No fundus reflex could be seen. The pupils were small and reacted slightly to light. The irides appeared normal. The anterior chambers were very shallow. The corneas were clear. Both globes may have been slightly smaller than normal. Intraocular pressure seemed normal to palpation.

There has been no feeding problem, and the baby has gained weight steadily. No microcephaly was noted. A congenital heart lesion was diagnosed (patent interventricular septum). The baby is now three months old and weighs 9 pounds and 2 ounces. The mother had German measles when four weeks pregnant.

Case 3. T. C., a girl born one week early without instruments, and weighing 4 pounds and 4 ounces had, since birth, a dense, white cataract in each eye. No fundus reflex could be seen. The eyes followed a light stimulus and exhibited a searching or nystagmoid motion. The pupils were 3 mm. in size and reacted sluggishly to light. The irides appeared normal. The anterior chambers were perhaps slightly more shallow than usual. The corneas were small and clear. There was definite microphthalmia of both eyes. The intraocular pressure was normal to palpation. Feeding the baby has been quite a problem, and now, at the age of five months, the weight is 8 pounds and 2 ounces. Microcephaly is present. There is a congenital heart lesion thought to be a patent ductus arteriosis. Other anomalies are mild pyloric stenosis and slight umbilical hernia. The mother had German measles during the first month of pregnancy.

Discussion

All three of the author's patients had congenital cataracts and congenital heart lesions. All mothers contracted German measles within the first month of pregnancy, which was during a rather severe epidemic of German measles in the East, about one and a half years ago. In the examination, the pupils in these cases were not dilated because the anterior chambers were quite shallow. It was thought that any additional information that might be obtained from mydriasis would not warrant the chances of inducing a glaucoma. Operations will be performed on these patients but they have been deferred for a month or so until the general physical condition stabilizes itself and a better evaluation of the heart condition can be obtained before the general anesthesia is given.

Some imponderables of this subject are: 1. Is the infection of the mother German measles?

2. If it is German measles, then why have we not noted before the occurrence of congenital anomalies in the children so infected?

3. If it is German measles, are the congenital anomalies which are now appearing the result of a more virulent type, or of an altered type, which may have gained access to this country through an increased traffic with Australia?

4. Has the cause-and-effect relation between other infections of the mother in the first three months of pregnancy and congenital anomalies existed in the past, but not been recognized?

5. Can any prophylactic measures be taken to prevent pregnant women from contracting German measles during the first three months of pregnancy?

6. Should pregnant women who contract exanthemata in the first three months of pregnancy be aborted?

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THE METABOLISM OF THE CORNEA*

Studies on the oxygen consumption of corneas of riboflavin- and vitamin-A–deficient rats

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The function of certain vitamin substances as prosthetic groups in respiratory enzymes is now firmly established. An insufficiency of one or of a group of these substances leads to definite pathologic changes in a variety of tissues, including those of the eye. Morphologic changes in the cornea have been cited as specific and pathognomonic of vitamin-A and riboflavin deficiencies. However, the associated metabolic disturbances of this particular tissue have not been studied.

Because of the intimate association between cellular activity and the uptake of oxygen, it is customary to use the measurement of the latter as one index to the rate of normal or altered metabolism. This fact rests on a firm basis, especially in the case of riboflavin, which, in the form of the flavoproteins (d-amino-acid oxidase, xanthine oxidase, cytochromereductase, and others) has an oxidation-reduction potential falling between the pyridine nucleotides and the cytochromes. The function of vitamin A as a prosthetic group in tissues other than the retina has not been shown, but since it obviously has some influence on epithelial structures it will be considered in a similar manner.

Duke-Elder[1] states that the metabolism of the cornea is extremely sluggish. Gundersen,[2] on the other hand, quoting Bessey's work, claimed that excised corneas of rats showed an astonishingly rapid metabolic rate.

Working with the Warburg respirometer, Bessey[3] found that the entire cornea utilizes approximately 4 cubic millimeters of oxygen per hour per milligram dry weight; that is, four sevenths as much as liver tissue, four sixths as much as muscle tissue, and four times as much a cartilage. Kohra,[4] measuring oxygen consumption of excised rats' corneas with the Warburg manometer, found a high metabolic rate for corneal epithelium. Although he had not demonstrated it experimentally, he nevertheless concluded that the endothelium also has a high metabolic activity. On the other hand, the parenchyma was found to have a low metabolic rate. Orzalesi,[5] repeating the same experiment, confirmed Kohra's findings.

In the present study, an attempt has been made to correlate the specific morphologic alterations in the cornea (resulting from vitamin deficiency) with changes in the associated respiratory rates. To this end the results are presented in two parts. Part I is a discussion of clinical observations of ocular changes in vitamin-A and riboflavin deficiencies. In part II we present data on the metabolism of the corneal tissues and indicate the possible significance of the findings in the light of associated anatomic changes.

Methods

The experimental animal

Approximately 200 albino rats from the Sprague-Dawly Institute strain were

* This study, aided by a grant from the John and Mary R. Markle Foundation, was undertaken by Dr. O. S. Lee, Jr., in partial fulfillment of the requirements for a Master's degree in Ophthalmology in the Graduate College, State University of Iowa.

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used as experimental animals. These rats were of both sexes and from 19 to 21 days of age when placed on the special diets.

Sixty rats were placed on a riboflavin-deficient diet and 25 rats on a vitamin-A–deficient diet. The remainder were fed on a control diet and allowed to grow to about 60 to 80 gm. in weight, when they were used for studies of normal corneas.

The rats were placed in individual cages with raised screen floors to prevent access to their own excreta. Tap water was given *ad libitum*.

THE DIET

A. Riboflavin-deficient diet. Two types of diet were used. Thirty rats were given Diet no. 1 and another 30 Diet no. 2.

Diet no. 1

	percent
Casein (vitamin free, Labco)	15
*Salt mixture (Hubbell no. 351)	2
Cod-liver oil	5
Sucrose	15
Corn starch (extracted with alcohol)	47
Crisco	14
Cellophane (ground)	2

One vitamine-B–complex pill of the following composition was given to each rat daily:

	mg.
†Thiamine chloride	0.04
†Nicotinic acid	0.20
‡Ryzamin B	50.0
Sufficient alcohol-extracted cornstarch to make pill mass.	

For the control diet, 0.04 mg. of riboflavin was added to the pill.

Diet no. 2

(For one kilogram diet, enough to last 20 rats 7 days)

Casein (vitamin free, Labco)	200.0 gm.
Sucrose	362.0 gm.
Cerelose	362.0 gm.
Salt mixture (Hubbell no. 351)	25.0 gm.
Cellophane (ground)	30.0 gm.
Cod-liver oil	20.0 gm.
†Choline chloride	1.0 gm.
†Thiamine chloride (Betabion)	3.0 mg.
†Pyridoxine hydrochloride (Hexabion)	3.0 mg.
†Pantothenic acid, Ca salt	10.0 mg.
†Nicotinic acid	50.0 mg.
†Alpha-tocopherol (ethyl laurate)	6.0 mg.
Para-aminobenzoic acid	100.0 mg.
Inositol	100.0 mg.

A week's supply was made at one time and kept in the ice box. If kept for longer periods or at room temperature, there is a reduction in thiamine potency.

In the control diet, riboflavin** (2 mg.) was added to the above mixture. Approximately five grams of food were eaten by each rat daily. The rats responded to the two riboflavin-deficient diets in practically the same manner. The second diet, however, was preferred because of its synthetic composition.

*B. Vitamin-A–deficient diet***

	percent
Casein (vitamin free, Labco)	20.0
Glucose	67.5
Salt mixture (Hubbell)	2.5
Cellophane (ground)	3.0
Spry (hydrogenated cotton-seed oil)	2.0
Brewers' yeast, Harris	5.0
Viosterol (Abbott's) 0.2 c.c. per kilogram	

PREPARATION OF CORNEAL TISSUES

A. Removal of cornea from the eyeball. The rats were killed by a blow to the occipital region. While the eyeball was held with a pair of fixation forceps, an initial incision was made by introducing a fine curved bistoury into the anterior chamber, after which the section of the cornea was completed with iris scissors. Care was taken to confine the cut to the limbus and not to include any scleral tissue. This precaution was necessary to

* Hubbell, R. B., Mendel, L. B., and Wakeman, A. J. A new salt mixture for use in experimental diets. Jour. Nutr., 1937, v. 14, pp. 273-285.

† Courtesy of Merck & Company.

‡ Burroughs Wellcome & Company.

** A modification of the vitamin-A–deficient diet for rats given in the U. S. Pharmacopeia, Eleventh Decennial Revised, p. 479.

prevent inclusion of iris and ciliary-body tissues with the cornea.

B. Removal of corneal epithelium. In experiments in which studies on the corneal epithelium and stroma were to be made separately, the epithelium was removed from the cornea of the intact eyeball by the following method: The globe was immobilized, and with a corneal spud the epithelium was scraped off carefully from the periphery to the center. With practice and reasonable care a complete sheet of epithelium was obtained by this method. This technique was checked by histologic studies and found to be satisfactory. After removal of the epithelium, the cornea was excised by the method previously described.

C. Removal of endothelium. An attempt was made to remove the endothelium for study but it was found impossible to obtain sufficient tissue for determination of oxygen uptake.

Both corneas from each rat were used in every experiment, and the tissues were placed at once in the respirometer cups which contain 0.5 c.c. modified Locke's solution.* In studies on isolated epithelial tissue it was found necessary to exercise care to keep the sheet of epithelium from folding on itself and thus preventing adequate contact with the substrate.

Measurement of oxygen consumption

Oxygen uptake was determined by use of six Barcroft micro-differential manometers which had been calibrated in the usual manner. Redistilled kerosene colored with Sudan III was used in the manometers. The manometer vessels were of approximately 5 cubic centimeters' capacity.

In all experiments each of the six vessels contained corneas or epithelial tissues from two eyes and 0.5 c.c. of the modified Locke's solution. The six compensating vessels contained enough of the same solution to make the fluid volume uniform. Each central well or cup of the respirometer vessels contained a roll of filter paper and approximately 0.1 c.c. of 20-percent potassium-hydroxide solution to absorb carbon dioxide.

The respirometers were mounted on a shaking device, similar to that described by Dixon,[6] having a rate of 60 cycles per minute with an amplitude of 2.5 cm. in a water bath whose temperature was 39° C. Measurement of oxygen uptake was begun at the end of a 20-minute equilibration period. Readings were then taken at 10-minute intervals for one hour. Results are given in cubic millimeters of oxygen (N.T.P.) per cornea.

The rate of oxygen uptake was invariably independent of the rate of shaking. When the manometers were gassed with oxygen, the results were not different from ungassed-manometer values. Therefore, diffusion was shown to be adequate with the technique used.

All calculations were made on the basis of corneal units rather than gram-weight units. This was done because of the very slight weight of the cornea and because this weight was quite constant (an average of 0.9 mg. dry weight per cornea for rats weighing around 70 grams). It is also obvious that the small mass of epithelium did not lend itself to accurate weighing. Since this is a comparative study between the metabolic activity of normal cornea and corneas of vitamin-A– and riboflavin-deficient rats

* The medium had the following composition (expressed as percent of the anhydrous salt)

NaCl	0.7
KCl	0.035
$CaCl_2$	0.025
$MgCl_2$	0.010
Na_2SO_4	0.025
glucose	0.075

Sorensen phosphate buffer (M/15) pH 7.4, 7.5 ml. per 100 ml.

rather than a comparison of oxygen consumption of cornea with other tissues, the present method of expression of results was considered to be adequate.

In order to permit a relatively accurate comparison between the oxygen uptake of normal and pathologic corneas on a unit-cornea basis, rats with normal corneas were taken for study when approximately 70 grams in weight. This weight was found to be similar to that of rats on vitamin-A– or riboflavin-deficient diets when they developed pathologic changes in the cornea.

Results

Part I. Clinical observations of ocular changes

Riboflavin-deficient rats

Ocular signs of deficiency were first observed after five to seven weeks. The palpebral fissures became narrowed and the eyeballs appeared sunken in the orbits. The lids were edematous and there was a loss of hair about the lid margins. These changes were followed soon by the appearance of a serosanguineous fluid in the conjunctival sac, which resulted in crusting of the lid margins and at times in complete sealing of the lids. The initial corneal change was not vascularization as observed by Bessey and Wolbach,[3] Johnson and Eckardt,[7] and others, but rather a fine, sandy roughening of the epithelial surface demonstrable with focal illumination. After staining with 1-percent fluorescein solution, observation with a loupe or corneal microscope revealed fine stippling of the corneal surface. This usually occurred after the seventh or eighth week, and was accompanied by engorgement of the limbic vascular plexus. After an additional 7 to 14 days, beginning vascularization of the cornea was observed. The appearance of vascular loops and the manner of their extension into the corneal stroma compared with the excellent and detailed description given by Bessey and Wolbach.[3] Gross corneal ulcers were noted in approximately 10 percent of the rats with vascularization of the cornea; the ulcers were superficial, perforation did not occur, and there was no massive sloughing of corneal tissue. Most of the animals were sacrificed for respiratory studies after corneal vascularization. In the few that were kept for further observation, the only additional corneal changes were an increased scarring of the tissue and some regression of the corneal vascularization. These animals usually did not live longer than two weeks after vascularization was first noticed.

Vitamin-A–deficient rats

The earliest manifestations were slight photophobia, lacrimation, and reddening of the conjunctiva which usually began during the third week of experiment. Shortly thereafter, the normally prominent protruding eyes appeared to recede into the orbit, with exaggeration of the aforementioned signs. A serosanguineous secretion developed. No changes were observed in the cornea until the fourth to sixth week, when a haziness was noticed, particularly about the periphery. The surface became dry, lusterless, and oily, and normal transparency was lost. The epithelial surface, however, remained intact, and no staining occurred with fluorescein. The degree and extent of the xerotic condition increased daily, and if the animal survived, keratomalacia usually developed within a week after the initial appearance of definite xerotic changes. With the appearance of keratomalacia, progression was extremely rapid, and in 24 to 48 hours, the cornea sloughed away in part or *in toto* and perforation followed. Examination of the corneas during the stages of xerosis failed to reveal the presence of vascularization. However, after the onset of kera-

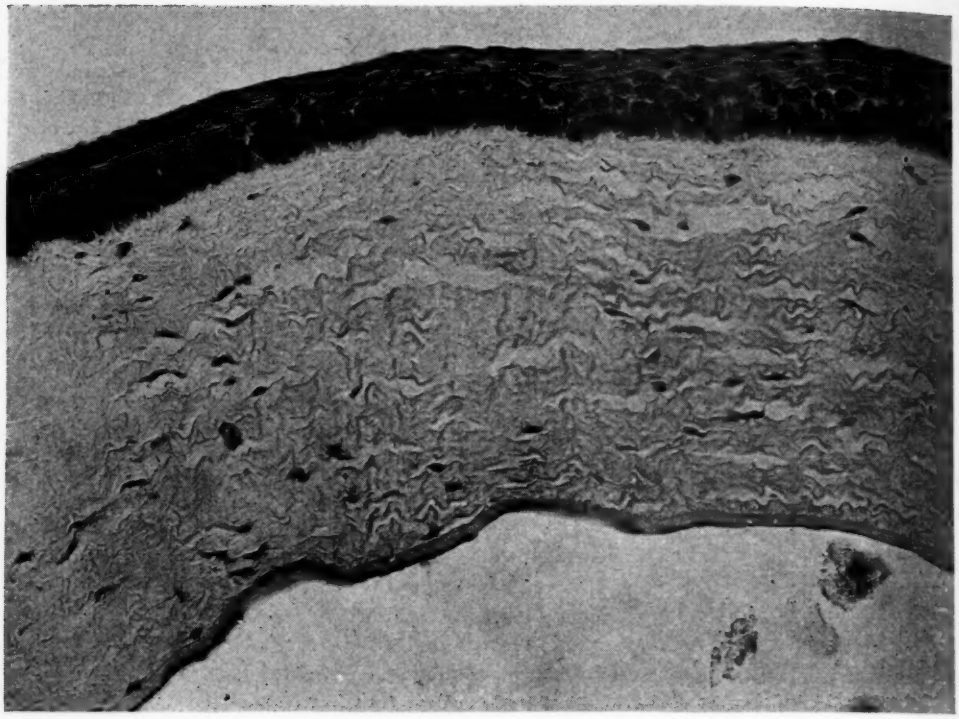

Fig. 1 (Lee and Hart). Normal rat cornea (×350).

tomalacia, vascularization was seen. A conspicuous feature of the pathologic changes was the remarkable vitality of the corneal epithelium, for it remained intact throughout the stages of xerosis. Even when necrosis of the stroma was taking place in beginning keratomalacia, the anterior epithelium was often preserved. This observation has been previously reported by Yudkin and Lambert.[8]

Part II. Metabolism of the corneal tissues

Normal cornea

The assumption sometimes made that the metabolism of the corneal epithelium is slow and contributes little to the metabolism of the intact cornea is not supported by the observations made in these studies, nor is it in accord with the observations of the rapid regeneration of corneal epithelium following injury. The results of the experiments here presented reveal that the oxygen uptake of the epithelium is rapid, for comparative studies show that the normal epithelium takes up the major portion of oxygen consumed by the excised intact normal cornea, whereas the stroma is relatively inactive (tables 1 and 2). The actual ratio of oxygen consumption between the stroma and epithelium is considerably greater than is shown in table 2, for these determinations were made on the basis of corneal units rather than on gram equivalents.

A comparison of table 1 with table 2 shows that the total oxygen consumption of the separated epithelium and stroma does not approximate that of the intact cornea. This discrepancy is to be expected, since there is some damage to the epithelial cells during the process of denudation. The technique used was at best relatively crude but still was superior to

other methods that were tried.

Since the endothelium consists of a single layer of cells on the posterior surface of the cornea it was found impossible to demonstrate its oxygen uptake with the present technique and instruments. Therefore, conclusions concerning the metabolic activity of the endothelium cannot be made at this time.

Cornea of riboflavin-deficient rats

In respiratory studies on vascularized corneas from riboflavin-deficient rats, it was found that the oxygen uptake of such corneas when intact was nearly the same as that of normal controls (table 3). Separate studies on the epithelium, however, showed its oxygen consumption to be markedly depressed, whereas the oxygen consumption of the vascularized stroma was much higher than that of normal corneal stroma (table 4). Further experiments revealed that if corneas were taken when epithelial damage only could be demonstrated and no pathologic change in the stroma was yet evident, the oxygen uptake of the epithelium continued to be low whereas the oxygen uptake by the stroma approximated that of the normal controls (table 5).

TABLE 1

Oxygen consumption of the intact normal cornea

Number of Experiment	Weight of Rat grams	Oxygen Consumption* 30 min.	60 min.
1	80	0.96	2.01
2	100	0.85	1.76
3	70	1.31	2.26
4	72	1.41	2.28
5	60	1.17	2.32
6	100	1.38	2.50
7	82	1.09	2.13
8	125	0.60	1.64
9	87	1.04	2.20
10	70	1.09	2.30
11	70	0.77	1.59
12	142	1.36	2.21
13	50	1.10	2.37
14	50	1.18	2.33
15	50	1.62	2.59
16	56	1.74	2.06
17	90	1.37	2.60
18	90	1.60	3.20
	Average	1.20	2.24

* Figures represent cu. mm. oxygen (N.P.T.) consumed per cornea.

Since histologic studies of corneas of riboflavin-deficient rats demonstrated that the first change to take place in the eyeball is swelling and necrosis of the corneal epithelium (fig. 2), it would be logical to infer that the decrease in oxygen

TABLE 2

Oxygen consumption of epithelium and stroma of normal cornea*

Number of Experiment	Weight of Rat grams	Epithelium 30 min.	60 min.	Stroma 30 min.	60 min.
1	100	0.38	0.97	0.27	0.36
2	100	0.28	0.61	0.18	0.34
3	80	0.63	1.21	0.41	0.68
4	80	0.42	1.71	0.20	0.30
5	70	0.78	1.56	0.05	0.10
6	72	0.62	1.22	0.41	0.60
7	100	0.61	1.20	0.46	0.56
8	102	0.59	1.18	0.40	0.67
9	140	0.60	1.13	0.32	0.47
10	68	0.55	1.10	0.35	0.52
11	92	0.49	1.03	0.03	0.05
12	70	0.49	1.00	0.23	0.73
	Average	0.54	1.16	0.28	0.44

* The corneal stroma also includes the endothelium which may be only slightly or almost completely stripped away.

TABLE 3

OXYGEN CONSUMPTION OF THE CORNEA IN RIBOFLAVIN DEFICIENCY. INTACT CORNEA WITH VASCULARIZATION

Number of Experiment	Weight of Rat grams	Oxygen Consumption 30 min.	60 min.
1	63	1.52	2.93
2	66	1.54	2.89
3	54	1.47	2.64
4	49	0.79	1.56
5	63	1.03	1.80
6	64	0.62	1.51
7	60	0.74	1.41
8	54	1.12	1.91
9	53	0.82	1.65
10	70	0.60	1.21
	Average	1.02	1.95

consumption of the epithelium was the result of the cellular destruction. The changes that follow damage to the epithelium consist of newly formed blood vessels which invade the stroma and are accompanied by cellular infiltrations (fig. 3). The presence of these cellular elements, which are absent in the normal avascular cornea, probably accounts for the increase in oxygen consumption observed in riboflavin-deficient corneas.

CORNEA OF VITAMIN-A–DEFICIENT RATS

In the experiments on xerotic corneas, the oxygen uptake of the entire corneas was the same or even higher than that of the normal controls (table 6). The oxygen utilization of the epithelium alone showed it to be in many cases higher than that of the normal corneal epithelium. The stroma, however, was found to be comparable to that of normal stroma in its relatively low metabolic activity (table 7). These findings are in accord with the results of Orzalesi,[5] who found the respiratory rate and glycolytic activity of vitamin-A–deficient corneas of rats to be more active than those of normal controls.

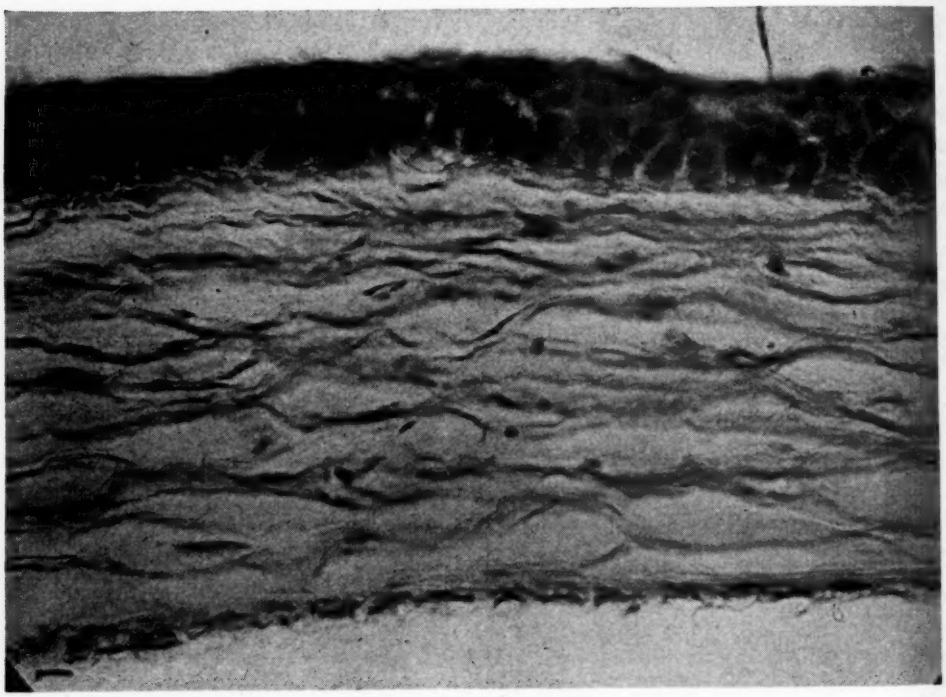

Fig. 2 (Lee and Hart). Cornea of riboflavin-deficient rat with early changes in the epithelium (×400).

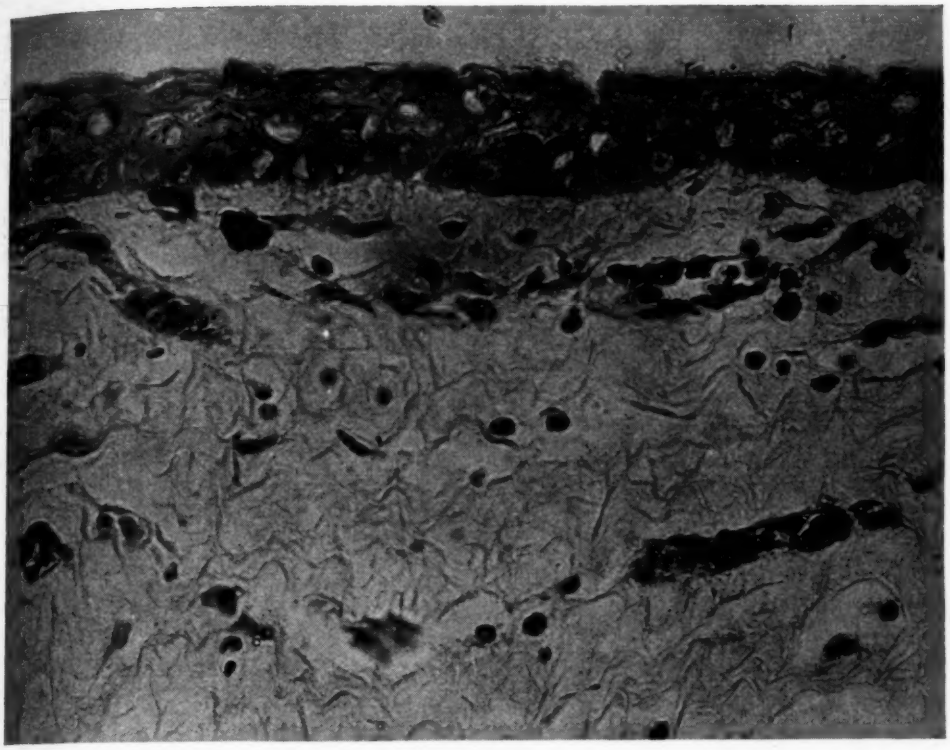

Fig. 3 (Lee and Hart). Cornea of riboflavin-deficient rat with more advanced changes in the epithelium and vascularization with infiltration in the stroma (×450).

He attributed this to the marked necrotic changes in the tissue which might free certain substances capable of accelerating metabolism.

The metaplasia and hyperlasia of the corneal epithelium represent the first manifest pathologic change in the eyeball of rats on a vitamin-A–deficient diet (fig. 4). Most of the corneas used in the present experiments were taken when the pathologic change was confined to the corneal epithelium and no changes were found macroscopically or microscopically in the deeper tissues. Therefore, the retention of approximately normal oxygen consumption of such corneas may be due to the absence of any damage to the vitality of the cells themselves, while even

TABLE 4

OXYGEN CONSUMPTION OF THE CORNEA IN RIBOFLAVIN DEFICIENCY. EPITHELIUM AND VASCULARIZED STROMA

Number of Experiment	Weight of Rat grams	Epithelium		Stroma	
		30 min.	60 min.	30 min.	60 min.
1	46	0.21	0.71	0.60	1.35
2	48	0.23	0.53	0.85	1.70
3	70	0.12	0.29	0.72	1.41
4	56	0.17	0.30	1.00	1.79
	Average	0.18	0.45	0.79	1.56

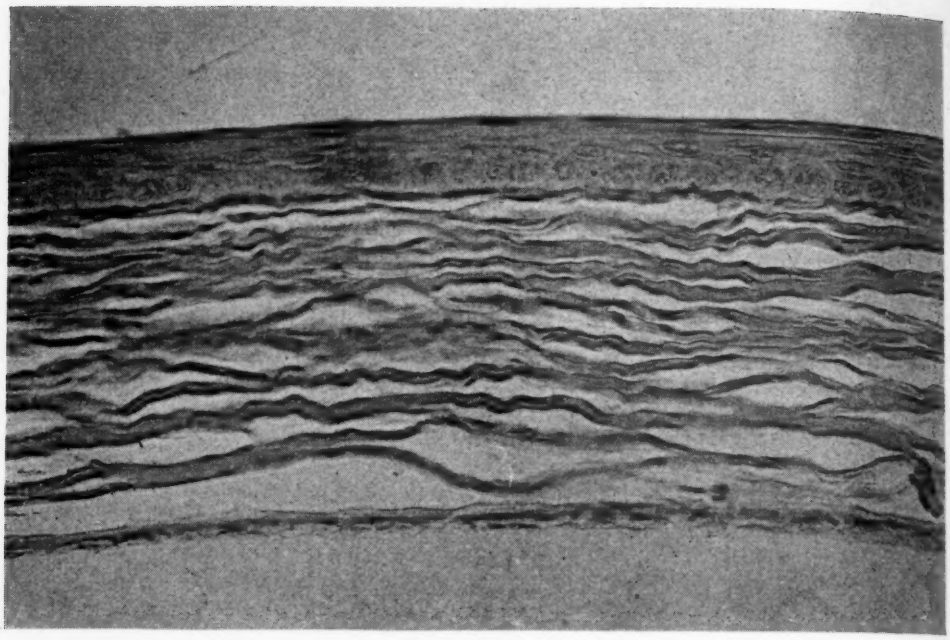

Fig. 4 (Lee and Hart). Cornea of vitamin-A–deficient rat with early changes in the epithelium (×400).

higher oxygen uptake could be ascribed to the cellular hyperplasia that has taken place in the epithelium. When more complete keratinization of the epithelium had occurred, vascularization of the stroma was observed. New blood-vessel formation usually is associated with necrosis and cellular infiltration of the stroma (fig. 5). These changes representing an advanced stage of the vitamin-A deficiency rapidly result in complete sloughing of the tissues and rupture of the cornea.

Figure 6 is a composite chart which gives a graphic view of all the data on oxygen consumption.

Comment

There has been no clear-cut demon-

TABLE 5

Oxygen consumption of the cornea in riboflavin deficiency. Epithelium and nonvascularized stroma*

Number of Experiment	Weight of Rat grams	Epithelium		Stroma	
		30 min.	60 min.	30 min.	60 min.
1	50	0.15	0.31	0.28	0.56
2	70	0.14	0.28	0.11	0.19
3	50	0.22	0.54	0.04	0.12
4	52	0.08	0.17	0.40	0.52
5	60	0.10	0.41	0.28	0.56
	Average	0.14	0.34	0.22	0.39

* In experiments in which nonvascularized corneas were used, only those eyes which showed a diffuse haziness of the epithelial surfaces were taken. These corneal changes represented the earliest manifestations observed in riboflavin deficiency of rats and consisted of edema and necrosis of the epithelial cells, as illustrated in figure 2.

stration of the sequence of changes occurring in the eye in the vitamin deficiencies in question. Indeed most authors have not seen the need for making such observations. Nevertheless, this consideration may have the utmost importance in the attempt to unravel the physiologic changes involved in vascularization. With regard to riboflavin deficiency in the rat, Bessey and Wolbach[3] state that capillaries grow into the cornea from the limbus after the fourth week. Quoting from their paper, "This happens before any change in the cornea visible in the gross has taken place. The transparency is undiminished. While these vessels may be seen by slitlamp illumination, no turbidity of the cornea or change in the corneal epithelium is revealed by this method or by histological study." And from the same paper with regard to vitamin-A deficiency: "We are disposed to disregard the inflammatory explanation of vascularization. Always, however, the presence of capillaries in the cornea was accompanied by a characteristic change in the corneal epithelium, indicative of the shift to a keratinizing

TABLE 6*

OXYGEN CONSUMPTION OF THE CORNEA IN VITAMIN-A DEFICIENCY. INTACT CORNEA

Number of Experiment	Weight of Rat grams	Oxygen Consumption 30 min.	60 min.
1	100	0.70	1.63
2	70	1.30	2.46
3	68	1.07	2.05
4	75	1.26	2.30
5	56	1.20	2.06
6	55	0.90	1.79
	Average	1.07	2.21

* The corneas used in these experiments were in various stages of xerosis. The xerotic process, however, was in all cases grossly visible to the unaided eye. A few of them showed beginning keratomalacic change in the stroma, as evidenced by an early necrosis of the stromal fibers on histologic study. However, no corneas with softening of the cornea visible macroscopically nor those in which perforation had taken place were used.

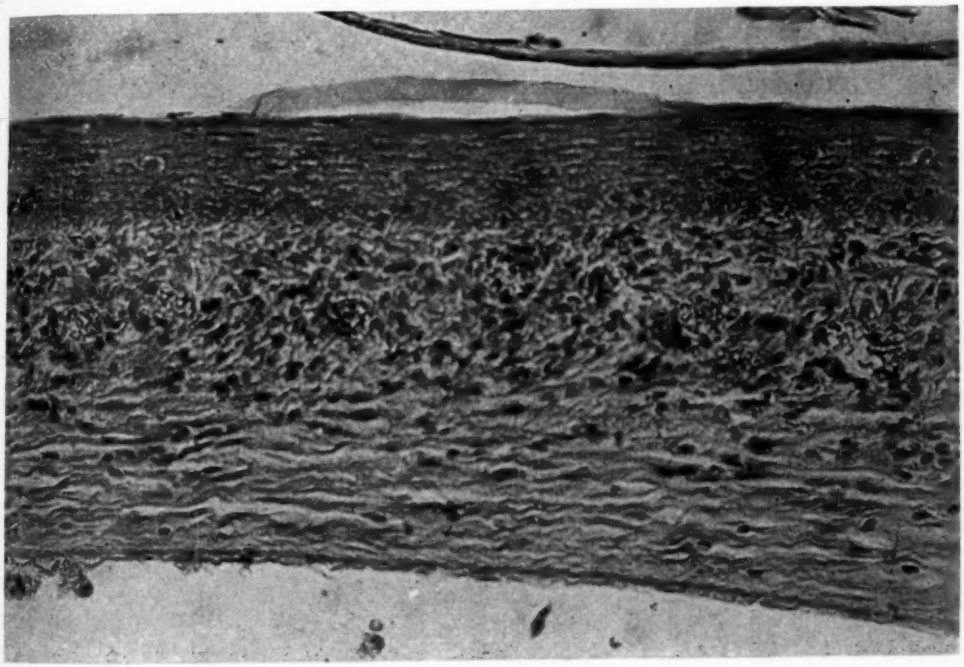

Fig. 5 (Lee and Hart). Cornea of vitamin-A–deficient rat with more advanced changes in the epithelium and vascularization with infiltration in the stroma (×400).

hyperplasia. The speculation is warranted that an important factor in causation may be a change in permeability of the epithelium in its effect upon respiration of the cornea as a whole."

These authors postulate a functional change rather than a change in the morphology of the epithelium to account for the ingrowth of vessels. Wolbach and Howe[9] had previously observed that in

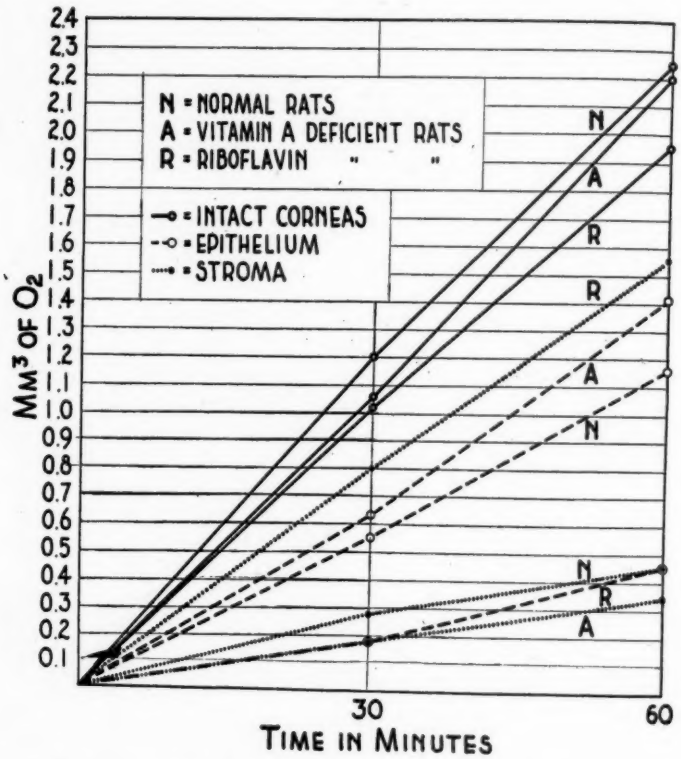

Fig. 6 (Lee and Hart). Composite chart from tables 1, 2, 3, 4, 6, 7, showing the average oxygen consumption of normal rat corneas and rat corneas of vitamin-A and riboflavin deficiencies.

TABLE 7*

OXYGEN CONSUMPTION OF THE CORNEA IN VITAMIN-A DEFICIENCY. CORNEAL EPITHELIUM AND STROMA

Number of Experiment	Weight of Rat grams	Epithelium		Stroma	
		30 min.	60 min.	30 min.	60 min.
1	85	0.35	0.80	0.17	0.26
2	65	0.86	2.01	0.21	0.39
3	60	0.84	1.95	0.16	0.30
4	70	0.60	1.50	0.14	0.31
5	60	0.40	0.90	0.20	0.32
6	50	0.56	1.05	0.17	0.40
7	75	0.72	1.50	0.20	0.35
	Average	0.62	1.42	0.18	0.33

* See footnote, table 6.

vitamin-A deficiency vascularization of the cornea followed keratinization of the epithelium and postulated that the response was due to greater physiologic demands from active and changed epithelium. In the later work, Bessey and Wolbach[3] reviewed the same material and, finding certain similarities to their observations in riboflavin deficiency, decided that vessel ingrowth occurred concurrently with the epithelial changes. This order of events apparently appeared to the authors to preclude an "inflammatory" explanation of vascularization. It would be difficult to criticize the inflammatory hypothesis since the mechanism of such a reaction is entirely unknown, nor is it altogether clear what is meant by a "change in permeability."

A variety of possibilities might be drawn upon in developing a logical explanation for vascularization of the cornea under these conditions. The suggestion offered by Bessey and Wolbach that vascularization is a response to asphyxia of the tunica propria has merit in that it suggests a new tack for further work, and indeed constituted the chief stimulus for the present endeavor.

At present we may safely assume some intimate relationship between the epithelium and the stroma, but the fundamental mechanisms are still obscure and no simple explanation can dispose of the problem. It is a significant fact that in our experiments, vascularization of the corneal stroma in both riboflavin- and vitamin-A–deficient rats is preceded by a morphologic change in the epithelium.[3]

Summary

1. The normal epithelium of the cornea is shown to have a high oxygen uptake in contrast to a low uptake by the stroma. Because the endothelium consists of only a single layer of cells, it was impossible to measure its oxygen consumption by this method.

2. The oxygen uptake of the corneal epithelium of riboflavin-deficient rats was found to be lessened. This may be accounted for by the cellular necrosis present in the epithelial cells of such corneas. When vascularization was present, the oxygen uptake by the stroma was found to be elevated, whereas in the absence of vascularization the oxygen uptake remained low. It is reasonable to assume that the increased oxygen uptake by the stroma is the result of vascularization and cellular infiltration.

3. The metabolic activity of the xerotic cornea was found to be normal or higher than normal. The presence of metaplasia and hyperplasia instead of an actual cellular destruction of the corneal epithelium may be responsible for the approximately normal oxygen consumption of corneas of rats with vitamin-A deficiency.

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THE USE OF TYROTHRICIN, A BACTERIAL EXTRACT, IN THE TREATMENT OF MARGINAL ULCERS OF THE CORNEA*

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In 1939, Dubos[1] reported the isolation, from bacterial culture, of a substance that destroyed gram-positive cocci. This antibacterial extract was tyrothricin; it was prepared from cultures of the Bacillus brevis, an aerobic sporulating saprophyte commonly found in sewage and soil.[2, 3] Tyrothricin is a stable mixture of gramicidin and tyrocidine, both of which can be crystallized, with some difficulty, from the mother substance.[4] The antiseptic effect of tyrothricin is principally due to its content of gramicidin. Because of its greater stability and relative ease of preparation, tyrothricin is the substance preferred for clinical use.[5]

Gramicidin is a polypeptide that has been shown to inhibit the growth of gram-positive cocci.[5] Although this bacteriostatic action varies in effectiveness against different strains of these microörganisms, it is, in general, highly effective in even minute dosage of the drug.[6] Meningococci and gonococci are also inhibited by gramicidin, though to a lesser degree, whereas other gram-negative microörganisms have proved invulnerable to this substance.[5] It has been further shown that the presence of gram-negative bacilli interferes with the bacteriostatic action of gramicidin, so that it is quite ineffective against mixed infections. The drug is also inactivated by cephalin.[5] Gramicidin has been shown to be slightly hemolytic,[7] a property which can be inhibited by small amounts of glucose or mannitol.[5]

Tyrocidine, which comprises 80 percent of the tyrothricin mixture, is a protoplasmic poison equally effective *in vitro* against gram-positive and gram-negative organisms.[3] *In vivo*, however, its action is largely destroyed by serum; it therefore contributes little to the effectiveness of tyrothricin as an antiseptic. The hemolytic and leucocytolytic effects of tyrothricin in large dosage have been attributed to its content of tyrocidine.[7]

Tyrothricin is soluble in alcohol and acetone but not in water or body fluids. It is also very poorly diffusible. Because of these physical properties and the toxicity of its component tyrocidine, tyrothricin is not employed systemically.[7,8] However, its poor absorbability and highly potent bacteriostatic action in small dosage renders it a safe and very effective antiseptic against gram-positive organisms by topical administration or the irrigation of infected body cavities.[9]

The conjunctival sac presents a surface prone to infection with gram-positive organisms, especially staphylococci, streptococci, and pneumococci. Moreover, it is easily treated by topical application without danger to the body by absorption of the medication into the general circulation in any appreciable quantity. It was therefore thought that in infections of the ex-

* From the ophthalmological service of Dr. R. K. Lambert, The Mount Sinai Hospital.

ternal eye and associated structures a favorable opportunity is provided for the therapeutic trial of tyrothricin.

Because of the greater availability of the more widely used antiseptic agents, and their usual efficacy in the treatment of the common infective conjunctivitides, no attempt was made at this time to treat such cases with tyrothricin. However, several patients with stubbornly resistant marginal ulcerations of the cornea were brought to the hospital and these afforded an opportunity for a therapeutic test of this drug.

Marginal ulcers of the cornea are not uncommon. The most frequent type is that associated with the more severe catarrhal conjunctivitides. In these cases the keratitic complication is considered an extension of the septic process in the conjunctiva. Treatment of the conjunctivitis with any of the usual antiseptics, occasionally supplemented by cauterization of the corneal lesion, usually results in rapid healing of the ulcer.[10]

There is another group of marginal corneal ulcers, more common in old people, that occurs without apparent cause, and is not associated with conjunctival inflammation.[11] These arise as marginal, subepithelial infiltrates which coalesce and break down to form superficial erosions crescentic with the limbus. Local ciliary injection is usually present, but very little general conjunctival reaction occurs. Such superficial ulcers usually heal readily when antiseptic applications or cauterization and pressure dressings are employed. However, they not infrequently prove quite resistant to such measures, and persist with distressing symptoms of photophobia, lacrimation, and pain.

Four instances of superficial marginal ulcerations of the cornea, not associated with conjunctivitis, and resistant to the usual forms of therapy, were recently treated with tyrothricin at this institution. An alcoholic solution of the latter was suspended in distilled water in a concentration of 0.2 mg. per cubic centimeter. An ointment was also compounded consisting of tyrothricin in a petrolatum-lanolin base, in the same 1:5,000 concentration. Such extreme dilution had previously proved effective against infection in other areas,[9] and it eliminated the possibility of absorption in any quantity sufficient to cause toxic reactions. Instillation of these preparations in the eyes of rabbits and normal volunteers proved their innocuousness; subsequent therapeutic applications in numerous cases have failed to reveal any local or systemic toxic reaction.

Case 1. A. S., a man, aged 67 years, presented himself in the Out-Patient Department with the complaint of having had pain in the right eye for several days. Examination disclosed a staining marginal ulcer of the cornea of the right eye, with local ciliary injection. There was no evidence of associated conjunctivitis. Cultures of material taken from the corneal ulcer were reported to show the presence of Staphylococcus aureus A and Staphylococcus albus B. The left eye appeared to be normal.

A 1-percent solution of silver nitrate was applied to the lower lid of the right eye; a drop of 20-percent argyrol was prescribed to be instilled three times daily, and bichloride of mercury 1:5,000 to be instilled in the right eye each night. After five days on this regime the corneal ulcer was found to have increased in size crescentically. It was then cauterized with tincture of iodine and instead of the previously prescribed medication 5-percent sulfathiazole ointment was instilled three times daily. Five days later the ulcer was found to be larger, and the regional ciliary vessels were more engorged. A pressure

dressing was applied but after several days no evidence of healing was seen. Mercurochrome 1 percent was instilled into the eye three times daily for five days thereafter, with no satisfactory results. The actual cautery was applied to the ulcer, but there was no apparent reduction in the size of the lesion. For 10 days thereafter the patient was treated only with 5-percent sulfathiazole ointment instilled into the right eye three times daily. During this period the staining lesion persisted unchanged and the patient's pain, lacrimation, and photophobia were very distressing.

Since routine methods had failed, the patient was instructed to apply one drop of the tyrothricin suspension to the surface of his affected eye every three hours, and to discontinue all other medication. After three days of application it was found that the ulcer was much smaller, ciliary injection much less marked, and the patient was greatly improved symptomatically. On the ninth day after tyrothricin therapy had been instituted no further staining of the cornea was present and the patient was considered cured. The region of the erosion presented a translucent superficial scar with no ciliary injection.

Case 2. R. L., a housewife, aged 58 years, appeared in the clinic complaining of lacrimation and pain in both eyes for two days. Examination disclosed small marginal infiltrates just within the nasal limbus of the left eye and similar lesions temporally in the right. There was some local ciliary reaction but no general conjunctival injection. One-percent atropine was instilled in each eye and 5-percent sulfathiazole ointment was prescribed for application three times daily, to be followed by hot compresses. After five days of this regime the infiltrates in the cornea of the left eye had disappeared, but those in the right persisted. One drop of 20-percent argyrol was then advised for the right eye, to be instilled every four hours. After two weeks, the infiltration had involved the entire limbal circumference and marked circumcorneal hyperemia was noted. In addition, just within the limbus inferiorly, two staining superficial ulcers were seen. Cultures of material taken from these erosions yielded Staphylococcus albus B. Bichloride-of-mercury ointment 1:5,000 was instilled and a pressure dressing applied repeatedly. One week later the ulcerations were larger. Cauterization of these lesions was performed with tincture of iodine, and pressure dressings were applied on two occasions during the following week, but induced no improvement. Three days later the ulcerations had coalesced across the lower corneal margin in crescentic fashion. Trichloracetic-acid cauterization was done, 5-percent sulfathiazole ointment instilled, and pressure dressing applied again. Two days later no improvement was noted.

At this point, after six weeks of therapy with the aforementioned agents, tyrothricin was prescribed. One drop of the suspension was ordered applied to the affected eye every three hours and tyrothricin ointment placed in the conjunctival sac at night. Three days later a diminution in the marginal infiltration and the ciliary injection was noted. The staining lesions of the lower corneal margin were definitely smaller and symptomatic relief was marked. Improvement continued uninterruptedly, and after two weeks of tyrothricin administration no further ulcer remained, the submarginal infiltrates had disappeared, and circumcorneal injection was minimal. At the site of the ulcer there remained a depressed white scar with slight peripheral vascularization.

Case 3. L. C., a woman, aged 72 years,

had undergone several epilations for trichiasis subsequent to an old trachomatous process of both upper lids. She presented herself at the clinic complaining of unusual pain, lacrimation, and photophobia in the left eye. Examination disclosed a marginal crescentic staining ulceration with ciliary injection at that point but no evidence of conjunctivitis. No cilium that might be irritating the cornea could be found on the lids at this time. A drop of 1-percent atropine sulfate solution was administered, 5-percent sulfathiazole ointment instilled into the conjunctival sac, and a pressure dressing applied. Two days later the lesion was larger and the patient's symptoms worse. The patient was then given tyrothricin solution to be instilled in the dose of one drop every three hours and the tyrothricin ointment was ordered applied nightly. Symptomatic improvement was rapid, and three days later the area of the staining lesion was noticeably smaller. After eight days the patient was completely cured and only an epithelized scar remained at the site of the ulcer.

Case 4. A. K., 45 years of age, presented a history of recurrent marginal corneal ulcerations. These previously had responded in a few days to local antiseptic therapy. On this occasion a staining erosion of the lower corneal margin of the left eye, not associated with conjunctivitis, had resisted treatment with 20-percent argyrol, sulfathiazole ointment, and repeated pressure dressings for 10 days. Cultures of the ulcer were taken twice, but no growth occurred, possibly due to antiseptics previously used. One drop of the tyrothricin suspension was instilled into the conjunctival sac every three hours, and all other medication stopped. The ointment was not employed after one application because the patient thought it irritating. After three days of this regimen symptomatic improvement was notable; on the fifth day ciliary injection had disappeared, and no further staining of the lesion could be seen.

Comment

The etiology of these primary superficial ulcerations of the marginal cornea is unknown. Fuchs[12] in 1893 considered them as a complication of the gouty diathesis. Duke-Elder[11] associates some with rosacea, but believes that most cases originate in a remote septic focus with metastatic corneal involvement occurring through the marginal vascular plexus. Whatever the cause, it seems very probable that infection, either primary or secondary, may be a factor in the persistence of the lesion and one against which active treatment is indicated. Where the more easily available antiseptics prove futile, tyrothricin appears to be a very effective therapeutic agent in this condition.

Since tyrothricin is now commercially available, the range of its clinical applicability deserves investigation. Studies are now under way to determine its effectiveness in other diseases of the eye. Further trial will be necessary to confirm our favorable impression of this apparently safe and extremely effective new antibacterial agent.

Summary

1. Tyrothricin* of Dubos has proven an effective antiseptic agent against gram-positive organisms.

2. It may be readily and safely applied to the conjunctival sac in effective concentrations.

3. Four cases of resistant marginal ulcers of the cornea are described in which tyrothricin was employed with gratifying results.

* Tyrothricin is marketed by Sharp and Dohme, Philadelphia, Pennsylvania.

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EXOPHTHALMIC OPHTHALMOPLEGIA*

Report of a case with thyrotoxicosis of unusually long duration

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Although somewhat more than 100 cases of exophthalmic ophthalmoplegia have been reported,[1] the syndrome still remains a clinical rarity. The causes of the exophthalmos and of the ophthalmoplegia, and the relationship of these ocular dysfunctions to disturbances of thyroid metabolism, are still unclear. Autopsy reports of cases of exophthalmic ophthalmoplegia are very rare, and it is for the purpose of adding to the available data for eventual clarification of this interesting entity that the following case history with the findings of post-mortem examination is reported. The case is of further medical interest because of the unusually long duration of symptoms of thyrotoxicosis, and their variability.

Case history

A. E. S., an unmarried white man, 55 years of age, and a butcher by occupation, was admitted to the U. S. Marine Hospital on June 30, 1942, complaining chiefly of generalized weakness of four months' duration.

* From the United States Marine Hospital.

Family history was irrelevant; there was no knowledge of thyroid dysfunction or organic disease of the nervous system occurring in any close relative.

The *past history* included uncomplicated mumps, measles, chicken-pox, and scarlet fever in childhood; lymphangitis of the right forearm in 1918, resulting from infection of a laceration of the right hand; pneumonia in 1928, with uneventful recovery; and a cholecystectomy in 1932, performed because of gastrointestinal disturbances and roentgenologic evidence of a nonfunctioning gallbladder. There was no history of syphilis, or encephalitis, or cerebral trauma.

History of present illness. The existence of detailed records of medical examinations of the patient (records of examinations for insurance, for determination of disability claims, and for war-pension purposes) facilitated the accurate formulation of the clinical course, and the following data were derived from these records and from the patient's story.

In 1913, the patient first became aware of a "goiter," which manifested itself only

as a slightly uncomfortable, diffuse swelling of the suprasternal portion of the neck until 1916, at which time nervousness associated with emotional instability developed. In 1918, the patient was drafted into the United States Army, but the development of exertional dyspnea and palpitation rendered him unfit for military service and he received a medical discharge after less than three months' service. The discharge diagnosis was hyperthyroidism. At that time (1918) *both* lobes of the thyroid were moderately enlarged, but exophthalmos was not present. The heart was not enlarged, a soft apical systolic murmur being the only cardiovascular abnormality noted.

Following his discharge from the Army, the patient continued to be disturbed somewhat by nervousness, episodes of palpitation, and a tremor of the hands, and in July, 1921, he noticed moderate weakness of both arms associated with a sense of numbness in the fingers, and weakness of grip. In August, 1921, physical examination revealed symmetrical enlargement of *both lobes* of the thyroid gland. The heart was just within normal limits in size; a precordial heave and a palpable thrill at the apex were present. There was a loud, blowing, systolic murmur at the apex which was not transmitted. One examiner also described a very indistinct short presystolic apical murmur which, however, was not heard by other examiners. The pulse rate was 90 beats per minute, and the response to exercise was normal. The blood pressure was 135/95. Ptosis of the left upper lid and a left divergent strabismus were present. The left eye was more prominent than the right. The diagnosis made was exophthalmic goiter.

The patient's vision seemed to become impaired somewhat, and intermittent diplopia, headache, and blurring of vision developed in September, 1921. Physical examination in November, 1921, revealed exophthalmos, ptosis of the left upper lid, and a left divergent strabismus. The visual acuity was 20/20 in the right eye and 20/40 in the left eye. Examination of the fundus showed obliteration of the physiologic cups of the nerve heads, without definite papilledema, and engorgement of the retinal veins. The heart was definitely enlarged to clinical examination, but the blood pressure was normal (108/76), and there were no irregularities in rhythm and no murmurs. The muscles of the arms were soft and flabby. The patellar reflexes were exaggerated. Romberg's sign was not present, and the sensory status was normal. The face was rather expressionless and immobile. Wassermann tests on the blood and spinal fluid were reported as "doubtful positive" from a city laboratory, but as negative from a larger state health laboratory. The colloidal gold curve was 0000000000, and the cell content of the spinal fluid was normal. Urinalysis revealed no abnormalities. The basal metabolic rate was then plus 24 percent. The examiners at that time suggested that the patient's difficulties might be due to a basilar meningitis or a brain tumor.

Three months later (in February, 1922) because of persistence of weakness of grip, clumsiness of hands, and the development of some difficulty in walking which he attributed to weakness, the patient again sought medical advice. The positive findings of physical examination then were enlargement of the *left* lobe of the thyroid gland; hyperactive deep reflexes, particularly marked in the patellar reflexes; weakness of the hands and arms, with greatly impaired ability to dorsiflex the hands and elevate the arms; positive Babinski sign bilaterally; slight labial ataxia; exophthalmos; external strabismus; and right hyperphoria. The heart and lungs were considered normal. Re-

sults of a urinalysis, blood count, blood Wassermann test, and X-ray examination of the skull were not abnormal. The diagnosis suggested by the examiners at that time was amyotrophic lateral sclerosis, but it was not substantiated by the subsequent course.

The muscular weakness of the extremities apparently subsided spontaneously, but the enlargement of the thyroid, the exophthalmos, and the nervousness persisted. Examinations in 1924 and in 1925 indicated that the *left* lobe of the thyroid remained more enlarged than the right. Von Graefe's sign and Moebius's sign were present, in addition to the exophthalmos, but the eyeball movements were normal. The ptosis was not evident at that time.

In 1926, the eyes were reported *entirely normal* by a competent ophthalmologist; there was no exophthalmos, no ptosis, no strabismus, no abnormality in the fundus. The patient's basal metabolic rate that year ranged about plus 5 percent, and a note was made that there was no clinical evidence whatsoever to indicate thyrotoxicosis, and that examination of the nervous system was entirely negative for evidence of gross organic disease. By 1930, even the enlargement of the thyroid gland and the tremor had disappeared, and the patient considered himself in relatively good health, although exophoria of the left eye had developed.

In 1932, while the patient was recuperating from the cholecystectomy, ptosis of the right lid developed gradually over a period of about one month, and ptosis of the left lid developed two months later. The bilateral ptosis persisted, and the patient elevated his lids with adhesive tape strapped to the forehead. The exophthalmos recurred, and the *right* lobe of the thyroid gland became enlarged. Tremor and hyperactive deep reflexes were again noted. The presence of these features, plus tachycardia and dermographia, prompted the attending physicians to return to the diagnosis of hyperthyroidism.

In 1938, the patient noticed the recurrence of generalized weakness, but he remained ambulant. In 1940, transient blurring of vision and diplopia occurred and spontaneously subsided. Exertional dyspnea and palpitation again developed, but there was no orthopnea, ankle edema, or precordial pain.

Since February, 1942, the patient had noticed that his weakness was becoming more pronounced; his voice became weak and hoarse, and ingested food was sometimes regurgitated through the nose. There had been a gradual weight loss of 47 pounds since 1924. These difficulties persisted, and the patient presented himself for admission at the U. S. Marine Hospital on June 30, 1942.

PHYSICAL EXAMINATION. The patient was an elderly white male, fairly well developed but in a poor nutritional state, appearing chronically ill; height 65 inches, weight 115 pounds. Orientation in time, place, and person was accurate, and there was no discernible impairment of judgment or insight. Anxiety, general somatic hypermotility, and a coarse tremor of the fingers and tongue were apparent.

Ocular examination. Moderate bilateral exophthalmos and complete bilateral ophthalmoplegia externa (including the levators palpebrae superioris) were present (fig. 1). The distance from the external canthus to the apex of the corneal surface was 18 mm. on the right, and 19 mm. on the left, by gross measurement. (Exophthalmometric determinations were not available.) There was no eyeball motion whatsoever. Slight edema of the lower lids was evident. The patient elevated his upper lids with adhesive tape attached to the forehead, in order to counteract the

ptosis. Ophthalmoscopic examination of the fundi was negative, the discs, vessels, and retinas appearing normal. Pupillary reactions were normal. The visual acuity was 20/20 in each eye. Determination of perimetric field vision indicated a very slight contraction of the visual fields. The patient was not aware of the paralysis of eyeball motion.

Trunk. The neck veins were dilated but base. The rhythm was wholly irregular, auricular fibrillation, and there was a pulse deficit of 34 beats per minute (ventricular rate 154 per minute, pulse rate 120 per minute). The blood pressure was 148/80.

The abdomen was normal except for a long right rectus scar covering a small incisional hernia. The extremities, genitalia, and rectum were normal.

Fig. 1 (Fagin, Pagel, and Sand). Anterior and lateral photographs of the patient reported, demonstrating an enlargement of the thyroid, the ptosis, and the exophthalmos (partially masked by the ptosis).

did not fill from below. The *right lobe* of the thyroid was enlarged to the size of a lemon, displacing the trachea to the left, and felt soft and cystic. No *bruit* was audible over the thyroid. The lungs were clear to clinical examination. The heart was enlarged to the left and downward, the point of maximum intensity being in the 6th intercostal space at the anterior axillary line. The sounds were loud and forceful, and the aortic second sound was louder than the pulmonic second sound. A loud, long, blowing systolic murmur was audible maximally at the apex and was transmitted medially to the left border of the sternum, and upward to the

Neurologic examination. (a) Cranial nerves: The ophthalmoplegia externa has already been noted. Other cranial nerves were normal. The patient spoke with a hoarse voice but oral and laryngoscopic examination revealed no abnormality of the nasal, palatal, or laryngeal muscles or mucosae. The vocal cords appeared entirely normal. (b) Sensory status: normal. (c) Motor power: generalized weakness of mild degree without any paresis or paralysis (other than the oculomotor) was present. This weakness was not affected by an injection of prostigmin administered to rule out any element of myasthenia gravis. Gait was normal, ex-

cept as modified by the weakness. (d) Cerebellar function: normal. (e) Reflexes: The deep reflexes were equal and slightly hyperactive. Abdominal and cremasteric reflexes were normal. Plantar reflexes were normal on the left, but exhibited a Babinski type of response on the right which, however, was not constant.

Laboratory data. Urinalysis, blood counts, erythrocyte-sedimentation rate, blood-sugar, and nonprotein-nitrogen determinations were all entirely normal. The basal metabolic rate was plus 65 percent. Spinal puncture was productive of clear, colorless fluid under normal pressure and with normal dynamics, normal cell count, and protein content (25 mg. percent). The blood and spinal fluid gave negative Kahn reactions. Gastric analysis revealed absence of free hydrochloric acid, even after the injection of histamine. Roentgenograms of the skull revealed no significant abnormality.

Roentgenographic examination of the chest indicated slight cardiac enlargement as gauged by the cardio-thoracic ratio (transverse diameter of heart, 14.7 cm.; transverse diameter of chest 27.7 cm.). However, the contour of the heart and the aorta, and the lung fields were normal.

Clinical diagnoses. 1. Thyrotoxicosis. 2. Cardiac disease: (a) thyrotoxic; (b) enlarged heart; (c) auricular fibrillation; (d) II; (e) C. 3. Cystic adenoma of the right lobe of the thyroid gland. 4. Exophthalmic ophthalmoplegia. 5. Incisional hernia.

Course. In view of the rapid fibrillation and the generalized weakness, digitalis was administered, and effected a decrease in the ventricular rate to 86 beats per minute with a pulse deficit of 6 beats per minute. After digitalization, the basal metabolic rate was plus 32.5 percent. Lugol's solution was then administered daily for a 10-day period, the basal metabolic rate falling to plus 17 percent. Coincident with these decreases in the basal metabolism, the patient reported improvement in strength, decrease of anxiety, and disappearance of palpitation. With a view toward decreasing the strain on the patient's heart, thyroidectomy was considered advisable, despite the duration of symptoms, and an operation was performed under local anesthesia on July 31, 1942. The day following the operation, the patient exhibited collapse, dyspnea, dullness and absent breath sounds over the right lower lobe, and a low-grade fever (37.8°C.). These difficulties were attributed to a postoperative collapse of the right lower lobe, and bronchoscopic aspiration yielded one ounce of thick mucopurulent secretion. Following the aspiration, breath sounds were audible over the right lower lobe, but the patient continued progressively downhill, expiring on August 2, 1942, two days postoperatively.

PATHOLOGIC ANATOMY. *Thyroid.* The thyroid gland removed at operation evidenced enlargement of both lobes, more pronounced on the right side, and weighed 64 grams. The surface of the gland was nodular, and, on sectioning the gland, the nodules were found to vary from 0.5 to 1.5 cm. in diameter. Fresh hemorrhage had occurred into some nodules, while others had a colorless, almost translucent appearance. A small area of calcification was present in one nodule. The fibrous tissue about some of the nodules appeared quite thick.

Microscopic examination of thyroid sections indicated that the nodules, apparently by their expansive growth, had compressed the thyroid tissue at their margins into a thick capsule-like structure (fig. 2). The acini within the nodules varied considerably in size, but papillary proliferation of the epithelium was not evident. However, most of the epithelial cells were of the high columnar type. The colloid immediately next to the lining cells ex-

hibited vacuolization, but was otherwise homogeneous and heavily staining in type. In some nodules foci of degeneration were evident wherein the acini were widely separated by a lightly staining, almost acellular ground substance in which an occasional small nidus of thyroid cells was observed. The colloid material in degenerating portions of nodules was more lightly stained than other portions.*

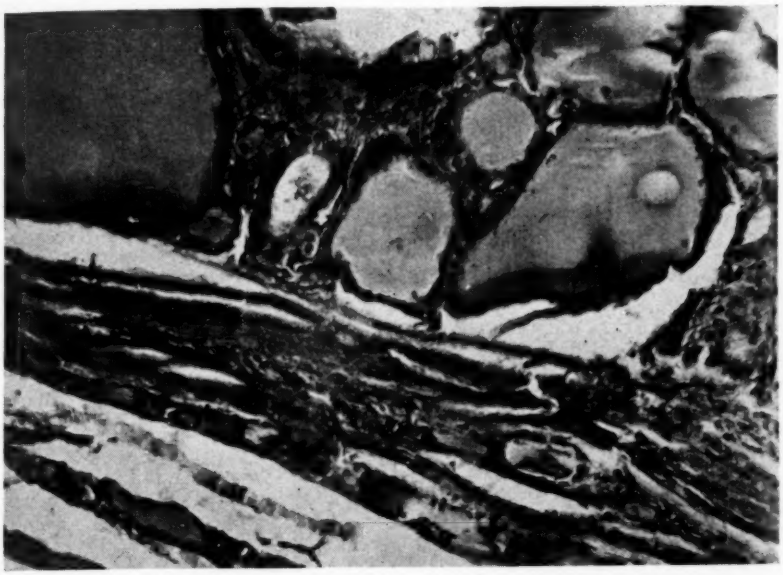

Fig. 2 (Fagin, Pagel, and Sand). Section through the thyroid gland (×100) demonstrating colloid retention, variability in size of acini, and compression of thyroid tissue to form a thick pseudocapsule.

POSTMORTEM EXAMINATION. *Gross anatomy.* The body was that of a middle-aged white male, 65 inches long and weighing 115 pounds. Postmortem rigidity and lividity were evident. Slight exophthalmos was present. Edentia, a recent operative scar of the neck, and a well-healed right rectus scar were noted on external examination. Superficial lymph nodes were not palpable. The body was opened by a Y-shaped incision. There was no increase in free fluid in the peritoneal, pleural, or pericardial cavities, and the serous surfaces were normal. The thymic fat pad was not enlarged.

The heart was dilated and enlarged, weighing 400 grams. All chambers appeared enlarged, but the left auricle was tremendously dilated. The myocardium was firm, red-brown in color, and showed no scarring. Slight atherosclerosis of the mitral valve and of the aorta was evident, but the coronary arteries were normal. The right ventricular wall measured 4 mm. in thickness, the left measured 13 mm.; the right auricle measured 3 mm., the left 4 mm. The tricuspid ring was 12 cm. in circumference, the pulmonic ring 7.5 cm., the mitral ring 10 cm., and the aortic ring 8 cm. Microscopic examination of the heart was normal except for the deposition of a moderate amount of yellow granular pigment at the poles of the nuclei. Slight atherosclerosis and intimal fibrosis were seen in sections of the aorta.

The right lung weighed 850 grams, and the left 950 grams. The lower lobes of both lungs (particularly the right) were almost completely consolidated, and the small areas which were not consolidated were extremely congested. The other lobes were crepitant and of the usual gray-red color lightly mottled with black. The bronchi were congested and contained thick

* Professor H. M. Trumbull in his report on the examination of five thyroid glands removed from patients in Brain's series states that these glands exhibited more evidence of colloid retention and less evidence of colloid secretion or transference than goiters characteristic of Graves's disease. The thyroid from our case would also fit into this group.

mucoid material. On microscopic examination, the alveoli showed varying degrees of polymorphonuclear leucocytic infiltration and minimal amounts of fibrin. Many of the alveoli in addition showed the lightly staining albuminous precipitate from edema fluid. The areas of consolidation varied in size and were mixed with areas of hemorrhage. The bronchioles were filled with purulent material.

Grossly, the other viscera were normal, except for slight enlargement and increase in the consistency of the liver.

hemorrhage into the perivascular spaces. The pituitary gland was normal microscopically.

The right eye was removed from the orbit, and the orbital fat was found to be firm and faintly lobulated. The external ocular muscles were thin and pale. Microscopic examination of sections of the orbital fat revealed no abnormalities. The external ocular muscles showed varying degrees of degeneration. A hyaline or Zenker type of degeneration was the most common type found. In the degenerated areas, the cross-striations and longitudinal striations

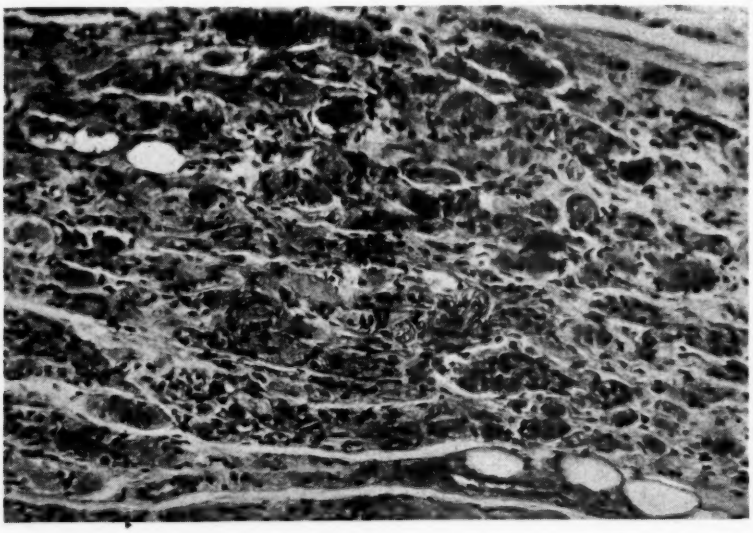

Fig. 3 (Fagin, Pagel, and Sand). Section through an external ocular muscle (×100) from the case reported. The hyaline degeneration and delicate fibrosis are evident. Compare with figure 4.

Microscopically, the liver revealed irregular congestion; other organs, including the thymus, were normal.

The vessels at the base of the brain exhibited a slight degree of arteriosclerosis, and the leptomeninges showed pronounced fibrosis, with hyperemia of the anterior two thirds of the leptomeninges. The Pacchionian granulations extended more laterally than usual. The lateral ventricle and third ventricle were slightly dilated, and the foramen of Monro was widely patent. A few petechial hemorrhages were seen in the anterior portions of the frontal lobes. On the right side there was destruction of a portion of the gyrus hippocampi involving the horn of Ammon. No other focal lesions were found in the cerebral hemispheres. Intensive examination of sections through the brain stem failed to reveal any significant abnormality. The pituitary gland was of normal size. Microscopic examination of the brain revealed only an occasional recent

were no longer visible, and in many places the fibers had degenerated to a round or oval hyaline mass with 3 to 6 nuclei at the margin. These masses took the stain more darkly than did the few normal fibers that remained. Occasional foci of lymphocytic infiltration were scattered through the external ocular muscles, and in the degenerated portions of the muscles there was a delicate and diffuse fibrosis enclosing the many small hyaline masses (figs. 3, 4).

ANATOMIC DIAGNOSES. 1. Nodular colloid goiter. 2. Enlargement and dilatation of the heart. 3. Bronchopneumonia. 4. Exophthalmos, bilateral, slight. 5. Passive congestion of the liver. 6. Degeneration and fibrosis of the external ocular muscles. 7. Emaciation.

Discussion and Comment

The case history presented is of equal interest to the internist and ophthalmologist. The long duration of the symptoms of hyperthyroidism and the variability of the symptoms are unusual. Apparently, the symptoms of hyperthyroidism first developed in 1916, subsided by 1926, recurred in 1932, and persisted until death. It is difficult to understand why the patient was not subjected to thyroidectomy earlier in the active phases of his disease.

The variability in the enlargement of the thyroid is another interesting feature in this history. Apparently, the first manifestation of dysfunction was a diffuse swelling of the gland, followed later (1922) by enlargement of the *left* lobe of the thyroid gland. Subsequently the enlargement became symmetrical, but in 1932 with the recurrence of the symptoms of thyrotoxicosis the *right* lobe of the gland became enlarged. The latent periods were apparently spontaneous in development since there was no history that the patient had taken iodine or other medications at any time previous to his admission to the U. S. Marine Hospital.

The variability in the ocular signs is another unusual feature of this case. It is not certain when the ophthalmoplegia developed, since the patient was not aware that he suffered therefrom. However, it is likely that paralysis of the external ocular muscles began to develop in 1940, with the transient diplopia, which was probably secondary to an initial asymmetrical paralysis.

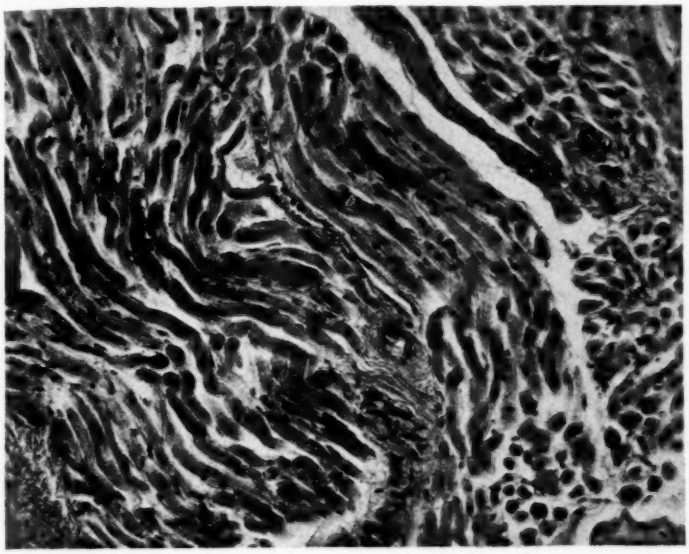

Fig. 4 (Fagin, Pagel, and Sand). Section through a normal external ocular muscle (×100).

The term *"exophthalmic ophthalmoplegia"* is descriptive of the clinical features of this entity; that is, protrusion of the eyeball in association with paresis or paralysis of the ocular muscles. The ophthalmoplegia is chronic, generally progressive, and is of the *externa* type, involving only the external ocular muscles. The causes of chronic ophthalmoplegia externa have been reviewed recently[2] and they fall into the following etiologic categories: 1. Cranial trauma. 2. Intracranial

and systemic infections. 3. Increased intracranial pressure. 4. Cerebral circulatory disorders. 5. Poisonings and miscellaneous causes. Exophthalmic ophthalmoplegia is included in the poorly defined miscellaneous group since its cause is unclear.

Brain and Trumbull,[3] in a comprehensive review, discuss the clinical and pathologic aspects of exophthalmic ophthalmoplegia from personal observation of 31 cases. Briefly, the exophthalmos and ophthalmoplegia may develop in one or both eyes, the protrusion being more often asymmetrical, either spontaneously during a period averaging about three to four months in a patient with no or minimal constitutional signs of thyrotoxicosis, or within a few days to two years after subtotal thyroidectomy performed on thyrotoxic individuals.

During the development of the oculomotor paralysis, the patient may notice diplopia as a result of asymmetrical involvement. The exophthalmos of exophthalmic ophthalmoplegia is usually of greater degree (averaging 7 mm.) than that encountered in hyperthyroidism (averaging 2.5 mm.) as gauged by exophthalmometric observations. Some edema of the lids generally accompanies the exophthalmos and even papilledema may develop. Unilateral or bilateral ptosis was present in about 25 percent of Brain's patients. The manifestations of thyrotoxicosis (palpitation, nervousness, weight loss, enlargement of the thyroid gland, tremor, tachycardia, elevation of basal metabolic rate, and other features) are usually mild. The prognosis is generally poor. The exophthalmos and ophthalmoplegia attain a maximum in a few months, and then remain stationary or regress only partially, with resultant disfigurement and limitation of ocular function. Treatment by thyroidectomy, irradiation of the thyroid and pituitary glands, ovarian hormones, or thyroid extract is unsatisfactory in the great majority of instances. For cases exhibiting extreme exophthalmos, Naffziger's orbital decompression operation may be helpful.

The relationship between exophthalmic ophthalmoplegia and the state of function of the thyroid gland is confusing. The evidence *against* any direct causal relationship between thyroid hyperactivity and exophthalmic ophthalmoplegia is impressive, and may be summarized as follows: (a) Exophthalmos alone, or exophthalmos associated with ophthalmoplegia, may develop or increase[1,3] following thyroidectomy, when the patient clinically is in a *hypothyroid* state, and the basal metabolic rate is normal or subnormal. (b) The symptoms of hyperthyroidism found in association with exophthalmic ophthalmoplegia are usually mild in degree. (c) The administration of thyroid extract or of thyroxine to patients with obesity, myxedema, or goiter very rarely produces exophthalmos.[4] In those rare cases where it does develop, the general picture is one closely similar to exophthalmic goiter, so that the exophthalmos cannot be attributed directly to the thyroid extract. (d) The exophthalmos of exophthalmic goiter may precede the constitutional symptoms of thyrotoxicosis, and even if the hyperthyroid state is terminated by extirpation of the gland or by spontaneous subsidence, the exophthalmos generally persists and may become even more severe. (e) The age and sex incidence of exophthalmic goiter and of exophthalmic ophthalmoplegia are different.[3] Exophthalmic goiter affects females more frequently than it does males—in the ratio of 9 to 1; in exophthalmic ophthalmoplegia, this female preponderance does not exist. The average age of patients with exophthalmic goiter is in the middle thirties, that of patients

with exophthalmic ophthalmoplegia is in the late forties. (f) Smelser[5] reported that the injection of anterior pituitary extract (containing thyrotropic factor) produced exophthalmos regularly in thyroidectomized guinea pigs. Friedgood[6] also found that exophthalmos was more readily produced by anterior pituitary extracts when the guinea pigs were in a state of thyroid deficiency. Similarly, Marine[7] found that exophthalmos was more readily produced in rabbits by methyl cyanide after thyroidectomy.

These findings militate rather strongly against ascribing any direct causal relationship to the thyroid in exophthalmic ophthalmoplegia. The problem is complicated by the complexity of the interrelationships between the endocrine glands. It has been shown in experimental animals that exophthalmos can be produced by the injection of anterior pituitary extract[5, 6, 7] even in thyroidectomized animals. Whether human exophthalmos is due to an increased secretion of the thyrotropic factor of the anterior pituitary hormone, or to a decreased secretion of active antithyrotropic factor, or perhaps to both, is not clear. Marine[7] has suggested that exophthalmos (in guinea pigs, at least) may be due to excessive anterior pituitary secretion acting directly on a sympathetic center in the hypothalamus, or acting indirectly through the gonads on that center. However, that theory does not appear tenable in the light of Smelser's finding[5] that removal of the cervical sympathetic ganglion does not prevent the development of exophthalmos in guinea pigs. Another difficulty is that excessive thyrotropic activity has not been demonstrated in the blood or urine of patients with thyrotoxicosis.[8]

A significant addition to the study of exophthalmos was made by Smelser's observations of the orbital tissues.[5] He found that in both experimental and human exophthalmos the orbital tissue was excessive in amount, the retrobulbar connective tissue, the fat, and the external ocular muscles being edematous and infiltrated with wandering cells. The exophthalmos was attributed to the mechanical effect of the increased orbital tissue. We were unable to demonstrate edema of the orbital tissues or of the external ocular muscles in the case reported here.

Brain and Trumbull[3] ascribe the exophthalmos to an increase in pressure in the fat-containing cone formed by the globe and the external ocular muscles, the posterior surface of the globe forming the base, the muscles forming the sides, the muscular attachments to the bony orbit forming the apex. The increased pressure presumably results in mechanical restriction of the motion of the eyeball, and interference with venous drainage results in edema of the muscles and orbital tissues. McGregor[1] postulates that cellular infiltration of the orbital tissues then occurs, which, together with the edema, results in an increased volume of orbital contents, with subsequent degeneration of the muscles. That such a hypothesis, although teleologically satisfactory, is not completely adequate is indicated by its failure to explain the ophthalmoplegia in such cases as that reported by McGregor himself, wherein the paralysis preceded the exophthalmos by three months.

A somewhat different explanation is tendered by Galli-Mainini,[8] based on the probability that there is a tendency toward the development of edema in hyperthyroidism (resulting from increased pulse pressure, subnormal serum-protein levels, capillary dilatation, and increased capillary permeability). These *edematogenic* factors, acting with myasthenia of the oculomotor muscles, result in an increase of volume and pressure in the orbit, and exophthalmos. Degeneration and lymphocytic infiltration of the extra-

ocular muscles presumably causes the ophthalmoplegia.

The treatment of exophthalmic ophthalmoplegia is a difficult clinical problem. It has been stated that thyroidectomy is the measure most definitely contraindicated in Graves's disease with dissociation of thyrotoxicosis and ophthalmopathy,[9] and the treatment recommended is to lower the basal metabolic rate with iodine to as low as it will go and then add thyroid hormone. Means and co-workers[9] point out that the metabolic action of the thyroid and the iodine will cancel each other, but it is the diuretic effect of thyroid that is desired in order to reduce orbital swelling. In the patient presented in this report, the thyroidectomy was considered advisable because of the cardiac factor; however, in retrospect, in view of the good response to the administration of digitalis and iodine, perhaps it would have been wiser to have continued those measures, and, if necessary, to have irradiated the thyroid later.

Summary

1. The clinical history and findings of postmortem examination of a patient with exophthalmic ophthalmoplegia are reported.

2. The syndrome of exophthalmic ophthalmoplegia is briefly reviewed.

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DUTIES AND TRAINING OF AN ORTHOPTIC TECHNICIAN

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It is clear that before planning a suitable course of study and training for an orthoptist we must come to some agreement as to the proper functions or duties of an orthoptist. It seems to me that the proper or specific sphere of the orthoptist is the dealing with disturbances of binocular vision and ocular motility and the teaching and training of the patient in the proper use of the two eyes by means of special exercises—"Reëducation of the brain to use the two eyes together," to quote Miss Billinghurst. It may be a matter of convenience to add other duties to this, her proper field, but for the purposes of fruitful discussion these supernumerary activities should be omitted. I have no quarrel with those who wish their flutist to double in brass and who therefore seek an orthoptist who is a perimetrist, a diagnostician, or even a refractionist or an expert in dealing with reading difficulties or an office nurse. What I am insisting upon is that these are not a part of the proper, or specific, exclusive sphere of the orthoptist.

In her capacity as an orthoptist it seems to be generally agreed that she should act in coöperation with, and under a certain amount of supervision by, an ophthalmologist. It is the proper or specific sphere of the ophthalmologist to make the diagnosis and plan and supervise the treatment. However, he can delegate certain details to assistants; one may measure the refraction, another may measure the motility. Just as the surgeon delegates the details of administering a general anesthetic to a specially trained anesthetist who is given rather a free hand, and as the laboratory technician is given a free hand in carrying out a requested blood test, so when the ophthalmologist decides that orthoptics is indicated he need not specify what in detail should be done by the orthoptist. If he is wise he will leave that to one who knows much better than he how to deal with the details. If the ophthalmologist makes any bluff of pretending to know, he may be sure the orthoptist will see through it very easily and he will be given a corresponding place in her estimation—she is usually a good diagnostician in that field.

But the ophthalmologist should weigh the data furnished him and plan the treatment, whether by wearing glasses, perhaps with prisms, by orthoptic exercises, by operation (and if so what operation and when), and by postoperative training.

From this it follows that diagnosis is not the function of the orthoptist except in a limited but very important way. This consists in making certain measurements of function or performance.

FUNCTIONS OF THE ORTHOPTIST

The orthoptist makes certain measurements:

(1) Visual acuity of each eye, separately and together, with and without glasses (bears on amblyopia, on alternating, on possibility of progress).

(2) Sometimes measures range of accommodation: Positive by −sph. and the near point of accommodation; negative by +sph.

(3) The deviation, whether phoria or tropia: A, by inspection (cover test and prisms); B, by instruments (amblyoscope, and other apparatus).

(4) Retinal correspondence and projection, normal or anomalous.

(5) Binocular vision analyzed according to the three established grades.

(6) Suppression must be investigated (very important).

(7) After determination of the deviation and fusion status, the amplitudes should be measured.

(8) Psychology: Patient's grade of intelligence, ability to coöperate, to concentrate. Patient's stage of growth (maturity); need of discipline; planning rewards. Mother: intelligence; coöperation; possibility of home treatment.

(9) The case must be analyzed and the problem clearly perceived as it exists at the present stage.

(10) Decide on the line of treatment. Have something definite in mind as the first goal. Work with precision toward the needs of the individual. Avoid haphazard fumbling. Avoid routine ruts. Avoid waste of time on what patient has already learned or what he is at present incapable of doing.

There are refinements of diagnosis important to the orthoptist but having little bearing on the treatment given by the ophthalmologist. For example, it is very important for the orthoptist to evaluate the patient's intelligence and actual capacity for coöperation; can he or, if he can, will he look at an object when told to? Some are not old enough to coöperate sufficiently to do this, some need discipline. Is the patient himself, or is his mother, capable of carrying out homework? If this is not diagnosed correctly much time and futile effort may be expended.

There are refinements of diagnosis important to the ophthalmologist but having little bearing on the treatment given by the orthoptist.

The ophthalmologist may wish to know the deviations as measured in the nine cardinal positions of gaze (often spoken of as six), though these rarely have any bearing on the work of the orthoptist who is satisfied to know the deviation in the primary position for distance and near and occasionally looking to the right, to the left, up, and down. I feel that much time is expended on irrelevant, unnecessary (though not uninteresting) measurements when the orthoptist might be much better employed otherwise.

What may the orthoptist expect of the ophthalmologist?

The ophthalmologist should report to the orthoptist:

(1) Findings with the ophthalmoscope, especially those bearing on amblyopia.

(2) Visual acuity, with and without correction.

(3) Refraction.

(4) Deviations, nature and etiology of deviations, if known. This is outside but close to the field of the orthoptist, who is not to be expected to be competent to investigate this problem but who may profit greatly from information which will help in making the prognosis, help her to answer the difficult question: How long will it take to cure this patient? (An important theme for research: Why do some get well in a few weeks while others, who appear similar at the start, take months?)

(5) History of treatment: Occlusion, glasses, orthoptics, operations, with details. Important information best furnished by the ophthalmologist rather than by the orthoptist by questioning the patient or mother.

(6) Conference or consultation, at least by telephone, at different stages of progress.

(7) Finally, the ophthalmologist should have sound and correct conceptions of the nature of the orthoptist's work. There are still many ophthalmologists who cling to the conception that disturbances of motility are due fundamentally to "muscle trouble"—some mus-

cle is weak or some muscle too strong. Inevitably they think of the orthoptist's work as trying to strengthen some weak muscle. The teacher of piano playing requires hours of practice playing scales and five-finger exercises, not to strengthen the muscles of the fingers and arms. The best player is not the one with the strongest muscles but the one who has the skill to use them. Proper use of the eyes is something that can be taught.*

What should be the relations between ophthalmologist and orthoptist?

(1) The ophthalmologist is in charge and entitled to dictate.

(2) Ideally, conference and consultation between the two should prevail, since each can contribute to solving the problems, each knows things the other does not know.

(3) When the ophthalmologist assumes the role of superiority the best way for the orthoptist is to assume the role of humility (and pity) but proceed, by deeds and by achievement impossible to the ophthalmologist, to show him the better way. Do not start by antagonizing him, leave him a chance to save his face.

(4) The ophthalmologist who would get the most out of the orthoptist should give her a free hand, offering suggestions in a friendly way, not in a dictatorial way.

(5) If there are differences of opinion, an attempt should be made to solve them by intelligence and discussion. Nothing is finally settled until it is correctly settled.

Having outlined the duties of the orthoptist, one is now prepared to take up the question of training or preparation of the orthoptist. As in the training of the ophthalmologist, it will be found that since many, very many things will be useful the chief problem is the relative importance or value of different subjects. Where should the emphasis be placed? Time devoted to this must be taken from that.

First of all, it is to be hoped that some consideration will be given to aptitude. Is the person of a type likely to be successful as an orthoptist? The mere fact that she is fond of children is not enough, this might do for a nursery maid. It requires a fairly high degree of mental equipment plus the training—preorthoptic and orthoptic—to make a good orthoptist. Mediocre intelligence will not shine in orthoptics.

Training of the orthoptist

What is the best preparatory (preorthoptic) course?

Training as a nurse, secretary, or laboratory technician does not offer the best basis for orthoptics. But the normal-school teacher, or one who has majored in psychology at college, or one experienced in kindergarten work has an advantage over even the graduate in optometry or in medicine.

Essentials of a course in orthoptics

Anatomy and Embryology. One can be a good orthoptist with only a smattering of anatomy of the ocular muscles and their blood and nerve supply—quite different from the ophthalmic surgeon. The orthoptist needs to know enough about the muscles to understand what the surgeon is trying to accomplish by his operations and to be able to give him good advice regarding operations. Thus she needs to know the different types of operation. She soon learns who among the surgeons get good results.

Physiology is very important, since orthoptics deals with functions and with

* See paper on "The role of exercises in ophthalmology." Trans. Amer. Acad. Ophth. and Otol., 1943.

reflexes in particular. A knowledge of the physiology of muscle, of nerve is important; also of the reflexes, conditioned reflexes. The secretions and the circulation are relatively unimportant. In physiology the instruction should be highly selective and specialized to fit the need of the orthoptist and give her a sound background so that she can understand the working of the visual organs, their possibilities, and their limitations. A smattering of *optics* (less even than that of the average ophthalmologist) is sufficient; also a smattering of lenses and prisms.

Physiologic optics. Training in this subject should include a smattering of *errors of refraction.* The student of orthoptics should know something of *accommodation,* especially the near reflex, which involves the accommodation, the pupil, and convergence. The study of *visual acuity* should comprise foveal and peripheral vision; the influence of age (infancy) on vision. A knowledge of *amblyopia, scotoma, suppression of small or large area of the retina* is recommended. *Binocular vision,* its three grades, should be studied: the states of development in infancy and childhood; corresponding and disparate points; fusion, motor and sensory.

The field of *ocular movements* should cover: monocular—fixation, reciprocal innervation; binocular—diplopia, fusion reflex, diplopiaphobia. Ocular muscles never work singly; effect of deficiency of a single muscle. All of the ocular muscles very strong, 100 times as strong as needed to move the eye. Innervation to ocular muscles always equal to both eyes. The two eyes act as a single organ, the binoculus.

Psychology is very important, especially applied psychology, covering growth and maturity; habit formation and control; learning (a large chapter); attention—capacity for concentration—influence of age; parents and home life; discipline—fears, rewards; application of teaching methods to orthoptics, especially elementary teaching—for example, methods of teaching spelling, reading, and so on; how to win the patient's confidence and coöperation—for example, by explaining the nature of the defects and the way the training achieves results and some of its limitations, and like factors.

Parents and intelligent patients are eager for such instruction and have a right to expect it. If the doctor does not give it (and he may not have the time) he should welcome the aid of the orthoptist in this important but much-neglected work. As a teacher she should be well fitted to teach the parents. If people are not taught by someone who knows, how are they to avoid the specious claims of charlatans or even of honest but ignorant practitioners? Moreover, patients crave understanding of their troubles. They wish to know why they are afflicted, what the causes are, what the remedies are, what the outlook will be. It is not easy to answer these questions when the questioner is as ignorant as most of them are. Hence some doctors fall back on easy evasions or sweeping generalities or old-time popular pathology, "The patient has a lazy eye or lazy muscle." Then if the orthoptist makes the matter clearer in plain language the patient or the mother thinks the orthoptist knows more than the ophthalmologist. Perhaps they are right.

Practice

The student should become familiar with: (1) the technique of the use of the instruments; (2) analyzing the visual problems; (3) planning treatment for specific problems; (4) discipline; (5) handling parents; (6) home work. It is not enough to tell the patient *what* to do —for example, bar-reading, which is often very difficult to accomplish correctly.

The influence of age is well illustrated by the progress of a child with convergence. He makes fairly good progress until he gets down to 10 degrees. There he often sticks discouragingly. He suppresses in spite of you, but when he learns to read he can be taught not to suppress and makes rapid progress.

(7) Reporting to doctors. The orthoptist has duties to satisfy the doctor, the parents, the patient.

The doctor wants to know if this case is one orthoptics will cure. He should (and would if he were wise) want the opinion of the orthoptist as to various matters of treatment including when to operate. He wants the patients to have confidence in him, hence the orthoptist must be very wary in her frequent conservations with the parents and the patients, striving to build up their confidence in the doctor, ready with explanations as to why operation is done or not done, "Trust the doctor, it is his job to decide such questions." "I have seen some splendid results with his cases." "He has had great experience in such cases." Unfortunately, patients are often keen enough to perceive that the doctor knows much less about his particular case than the orthoptist, who, of course, sees him 10 times to the doctor's one. However, some doctors prefer an orthoptist who does not know so much.

The orthoptist's duties to the parents cover:

(1) Explanation of the problem and of the treatment. The parents have more frequent contact with the orthoptist, become less afraid to ask her questions, and are very grateful for such information. To refuse it could have a bad effect on the relations between parent and orthoptist. (2) Instruction in home coöperation. (3) Satisfactory discipline.

The orthoptist's duties to the patient call for (1) understanding and (2) more understanding, (3) complete understanding. Factors are A, his age; B, his intelligence; C, his environment (note the marked change in attitude that comes soon after the child begins to go to school); how to manage motivation (rewards, praise, commendation, and the like as well as punishments); D, anatomic situation.

Summary

Ophthalmologists, in planning the proper course of study for an orthoptist, have over-emphasized certain parts of such subjects as anatomy, optics, some branches of physiologic optics; they have under-emphasized some aspects of physiologic optics, and especially of psychology, the science and art of teaching, the behavior of children, the fundamentals of learning, of acquiring new habits and eliminating old ones. It is easy to see why this should have happened since the doctors naturally stress the subjects they know something about and the subjects they know from experience are important in their own work. Knowing little about orthoptics they do not know what to emphasize.

Far be it from me to belittle the importance of the basic subjects, I have been pleading for them for years. It is the *relative* importance that I am advocating. All education involves frequent choices. I can take this only if I omit that. Hence the crucial decisions are: What can or must be omitted? I submit that in the training of orthoptists the failures have been not in the basic ophthalmic subjects but in the basic subjects of psychology, the art of teaching, the laws of learning, the ways of habit formation of conditioned reflexes.

Another thought is that perhaps orthoptics in this country has become inbred and needs crossing with new and different ideas to become most fertile.

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TREATMENT OF ASTHENOPIA—NONPATHOLOGIC AND NONREFRACTIVE IN ORIGIN

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A certain percentage of patients who consult ophthalmologists because of asthenopic symptoms are not relieved by glasses or other usual methods. Many of these give evidence of nothing that falls within the sphere of recognized diagnosis. A study of 125 of these difficult and dissatisfied individuals in our office leads us to believe that they manifest asthenopia from disturbed muscular coördination and/or fusional amplitude too small for classification, or spasm from muscles long held in restricted positions, passive congestion, and failure of smooth interaction of muscle and tendon, in relationship to adjacent tissues.

In our experience this group of 125 cases falls in a classification quite independent of the usual disturbances of coordination such as definite phorias and trophias, and they are seemingly all independent of refractive disturbance or intrinsic or surrounding pathologic factors. We believe there develops in many people a condition that disturbs the smooth, normal mechanical relationship of muscle and tendon and their coverings of tendon sheathes, Tenon's capsule, and the like, which produces various asthenopic symptoms. As muscles of the body generally become less pliable and interact with discomfort when not normally exercised, so the ocular muscles can, by assuming rather restricted action during long hours, become relatively fixed so that the movements less frequently required produce discomfort, as, for example, discomfort of the back muscles after bending over an operating table for prolonged periods.

Patients who do close work for long continuous periods sometimes are found to have in their eye muscles a condition analogous to the aforementioned backache. These muscles have been held in a converged, or other position so long that the eyes do not relax for an uncomfortably long period; or else they become comfortably fixed in these long-held positions and produce discomfort on being moved to less usual positions. Symptoms of our patients varied from constant or frequent headaches in 53 instances, to tearing, blurring, twitching, and general visual discomfort in the remaining 55 complete cases studied.

An occupational survey of the 44 men and 64 women patients reveals that 45 were confined to offices, 27 were students, and, of the remainder, 22 were housewives, 9 were professional people, 3 were salesmen, and 3 were employed in factories. The age range was from 12 to 54 years.

Uncorrected refractive errors and other visual irregularities, sinus disease, and like affections, which may be factors in asthenopia were ruled out insofar as it was possible to do so. All but eight patients attained visual acuity of 20/20 in each eye. The exceptions had acuity of at least 20/50 in each eye.

A specific diagnosis of the causes of the asthenopia found in this group of cases seems to us exceedingly difficult. Some of the patients had difficulty in abducting or adducting their eyes, hence they might be characterized as having "poor ductions," or a disproportionate relationship between the prism diopters of convergence or divergence, low "amplitude of divergence and convergence," or "insufficient amplitude of fusion areas." There is a little difficulty here in that too little agreement has been found as to defini-

tions for these terms. Nor is the relationship which should exist between factors such as abduction and adduction agreed upon. It is generally stated, however, that a "reasonable amplitude of convergence" is necessary to comfortable vision. We assume that some of our patients suffer from a lack of "reasonable amplitude of convergence."

Other diagnostic phrases that have also been suggested are "minimal binocular coördination," "disturbed binocular coordination," and "insufficient fusion span." But here again we find ourselves at a loss for lack of standards for measuring purposes.

We reach our decision as to the needs of the patient without benefit of special vocabulary. Our "technique" is most easily illustrated by a few examples.

A patient seated before the Synoptophore cannot hold two unlike figures superimposed at or near his phoria point. When he attempts to use his eyes simultaneously his fixation is poor. Asked to fuse identical objects he suppresses either alternately or monocularly. If "print jumps and blurs before the eyes after a short period of reading" and if headache develops in spite of refractive correction and other visual procedure, we try a series of exercises. Some persons suppress constantly when a load is put on, thus eliminating the necessity of coördination.

Another patient has no difficulty holding his eyes steady, but although he can abduct and adduct for what seems a reasonable degree he does so only with great effort and fatigue. His eyestrain if not otherwise relieved is frequently overcome with carefully administered exercises. Take such a patient's muscle balance with the phoropter and he may seem normal in the usual hurried examination, but hold him to it and he may manifest considerable muscular difficulty.

A third patient has difficulty adjusting accommodation and convergence quickly from one distance to another. He sees acutely after going through a period of conscious adjustment during which objects are blurred and occasionally double. A few exercises may make this adjustment more normal and comfortable.

As may be expected, a fair proportion of the cases classify themselves rather readily as convergence-insufficiency or -excess, or divergence-insufficiency or -excess problems. However, original phorias were normal in 80 instances, 15 patients were exophoric, and 13 were esophoric. No trophias were included. Original "duction" findings were poor in 79 instances, fair in 26 instances, and 4 patients had excellent ductions before beginning exercises. All but one patient had stereopsis, although with 13 patients this was of a very low grade.

At the conclusion of remedial exercises, phorias were found to be essentially unchanged. Improvement in "ductions" varied from 5 to 25 prism diopters. However, the conclusion that this improvement brought comfort does not, except in a few cases, seem warranted.

It is generally acknowledged that the ability to compensate for or to ignore weaknesses varies markedly in persons, and it is not far-fetched to assume that a systematic exercising of the various ocular muscles may result in a general improvement in the mobility of the eye muscles to the extent that the specific difficulty, if not incidentally corrected, may be more easily overcome.

We subjected each of these patients to a series of orthoptic exercises designed to correct the particular types of weaknesses toward which each showed a tendency and/or in general just to limber up the muscles and improve circulation. Many of the patients received base-in and base-out exercises sometimes combined with accommodation, alternation, or rotational

exercises. Some were given exercises especially designed to stimulate fusion by the foveal area; others received binocular peripheral stimulation. Exercises were given at both near and far points. Most of the work was done with a Rotoscope supplemented with the Synoptophore, the Tele-trainer and the Junior Metronoscope with prism attachment. The exercise period varied from 15 to 25 minutes, depending upon the type of exercise given. A trained technician remained constantly with each patient during an exercise. We believe constant, intelligent direction to be essential to effective orthoptics, particularly when it is desired to give a minimum number of exercises.

The number of exercises given to the patients varied from 3 to 21. However, the optimum number seemed to be 6 exercises, the first 4 being given bi-weekly, the last 2 at intervals of a week.

Home exercises were prescribed for two thirds of the patients. In seven instances these were the conventional stereoscope exercises. The other two exercises used were the well-known finger-to-nose convergence exercises and an equally simple divergence device familiar to our patients as the "thumb exercise."

Since this "thumb exercise" is flexible, simple to teach, and amazingly effective as a "relaxing" device, a brief description of it seems warranted. To perform the exercise, hold up the right thumb, 13 inches in front with good illumination at the back. Looking through to infinity will produce physiologic diplopia and two thumbs are seen out of focus. Now arrange the left thumb beside, parallel, and level 65 mm. from the first. Four thumbs will be seen with a little practice; one can fuse the middle two thumbs and by asymetric stereopsis project a third thumb. To exercise the lateral recti, gradually separate the two thumbs until the middle thumb is distorted against effort. The illusion will be aided and fixation stimulated if a spot is marked on each thumb nail with India ink about 2 mm. lateral to the midline. This spot will then appear to stand out in relief an inch closer than the thumb nail. Patients to whom this exercise is prescribed usually perform it several times a day for periods not exceeding one minute.

Relief from asthenopia was complete and thus far permanent (the program was begun two years ago) as concerns 108 of this group of patients. In several instances comfort followed many years of headaches and visual fatigue, in spite of glasses and other usual therapeutic procedures.

Of the 125 cases included in the study, 17 patients did not respond satisfactorily to remedial exercises. Various factors such as poor coöperation, absence from treatments, and illness were present in these instances and may have contributed to the lack of success.

As massage may give much relief in general to congested, stiff, and painful muscles anywhere in the body, so the orthoptic department of any ophthalmologist's practice can serve in addition a most helpful purpose over and above the more definite procedures for which such exercises are more commonly used.

Summary

A study was made of 125 cases of asthenopia in patients who were not relieved by glasses or other means. This group did not consist of those with definite muscular difficulties, such as definite convergence insufficiency and the like, but rather those who, for no apparent reason, had difficulty in doing sustained close work. The ages ranged from 12 to 54 years, with a mean age of 28. There were 44 men and 64 women in the completed cases; 27 were students, and of the remaining 37, 22 were housewives, 9 pro-

fessional people, 3 were salesmen, and 3 were employed in factories.

The vision in all but eight patients was 20/20 O.U., the others having at least 20/50 O.U. Orthoptic exercises varying from 3 to 21 sessions were given with the Synoptophore or other orthoptic instruments. Seventeen of the 125 failed to respond satisfactorily to remedial exercises. Poor coöperation and failure to take the optimum number of exercises, which was found to be six, may have been contributing factors.

It is thought that as massage may give much relief in general to congested, stiff, painful muscles anywhere in the body, so the orthoptic department in any ophthalmologist's office can serve in addition a most helpful purpose over and above the more definite procedures for which such exercises are more commonly used.

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NOTES, CASES, INSTRUMENTS

CAVERNOUS-SINUS THROMBOSIS WITH RECOVERY

Report of a case

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A soldier, 20 years old, was admitted to the Station Hospital on April 30, 1943, complaining of swelling of the nose and face, chills, and fever. Four days previously he had first noticed a small, red, painful swelling on the tip of his nose. This progressively increased to involve the upper half of the face at the time of hospitalization. Two days prior to admission he had had a shaking chill of 30 minutes' duration. Except for a mild sunburn no history of previous trauma or infection could be obtained.

Initial examination showed an acutely ill young, white, male complaining of painful swelling of the nose and face, the upper half of which was swollen, red, and tender. Although there was edema of the lids the eyes could be opened voluntarily, and there was no proptosis. The conjunctiva of the globes was moderately injected but not edematous. The pupils were round, equal, and reacted to light and accommodation. The eyegrounds appeared to be normal. Breathing was partially obstructed by the swollen nostrils, from which exuded a small amount of serous discharge. The neck was not rigid. Petechiae and rash were both absent. Physical examination was otherwise negative. His temperature was 104.2°F., the pulse rate 122, and respirations 34 per minute. The white-blood-cell count was 13,200, with 92 percent polymorphonuclears.

A diagnosis of cellulitis of the face originating from a lesion on the nose was made.

A blood culture was taken on the first day and 5 gm. of sodium sulfadiazine in distilled water administered intravenously. Sulfadiazine—2 gm. every four hours—was given orally. Continuous wet boric-acid dressings were applied locally.

The next day chemosis appeared and the edema of the lids became more marked. By the fourth day it was apparent that a bilateral cavernous-sinus thrombosis had developed. At this time the lids were so swollen that they could no longer be opened, and the ocular conjunctiva projected between the lids. Both eyes were proptosed and immobile. The supraorbital and palpebral vessels were markedly dilated. Fundus examination revealed engorged and tortuous veins. The optic discs were still well defined, with moderately

deep physiologic cupping. There were no retinal hemorrhages nor edema. Thick yellow pus was draining from three openings on the nose. The blood culture taken on the day of admission was reported positive for Staphylococcus aureus haemolyticus on May 3d, and sulfathiazole—2 gm. every six hours—was substituted for sulfadiazine. The patient became irrational and his temperature remained elevated. A white-blood-cell count of 20,000 was recorded at this time.

On the sixth day the patient was given a whole-blood transfusion (500 c.c. citrated). On the following day the swelling and induration has spread to the right mandible. Much of the ocular conjunctiva projected between the lids; drying was prevented by the use of a bland ointment and constant wet boric-acid dressings. On the same day he received 60,000 units of staphylococcus antitoxin (Lederle) intravenously and 40,000 on the following day. Twenty-four hours after the administration of the first of these treatments the patient's mental state improved and the temperature dropped to 101.4°F. From this day improvement was steady, with rapid recession of the facial edema. On the sixteenth day the dosage of sulfathiazole was reduced to 1 gm. every six hours and was discontinued three days later, after four days had passed in which the temperature was normal. The results of examination were now negative except for a residual distention of the palpebral vessels, which continued to improve, but was still present at the time of discharge on June 26, 1943. Vision was 20/20 in each eye, and the results of a fundus examination negative. Except for an occasional headache, the patient had no symptoms during convalescence.

Summary

A case has been presented of a 20-year-old soldier who developed cavernous-sinus thrombosis secondary to a pustule on the nose. Blood culture was positive for Staphylococcus aureus haemolyticus. During the first three days of hospitalization he received 5 gm. of sodium sulfadiazine intravenously and 36 gm. of sulfadiazine orally. After receiving the report on the blood culture sulfathiazole was substituted for sulfadiazine, and of this he received 116 gm. over a 16-day period.

The first signs of improvement appeared on the eighth day, 24 hours after an intravenous injection of 60,000 units of staphylococcus antitoxin. This was repeated with 40,000 units on the following day. The patient's condition, which had been critical up to this point, rapidly improved, so that at the time of discharge, on June 26, 1943, he showed only a mild residual distention of the palpebral vessels.

In view of the rapid improvement following the use of antitoxin one is tempted to attribute much of the good results to this therapy. Whether or not a favorable outcome would have been obtained if sulfonamides had not been used in conjunction with the antitoxin is uncertain. In such a critical condition one is prone to run the gamut of treatment and in so doing lose the ability to assess the value, individually, of the medications used.

Station Hospital.

BINASAL HEMIANOPIA

Irwin E. Gaynon
Lt. (MC), A.U.S.

Binasal hemianopia is that condition in which the nasal half of each field has no light perception. This type of field is rare, and is seldom of perfect bilateral division.

Frequency

Von Graefe (1858) described one case which he considered to be due to the

bizarre effects of a bilateral lesion of the occipital lobes. Mooren (1867) recorded one case as the result of hysteria. Daae (1869) reported six cases associated with optic atrophy in the same family which was considered to be an abiotrophy. Knapp in 1873 reported one case which he attributed to pressure of the sclerosed carotids on the optic chiasm, in a case of generalized arteriosclerosis. Herschel (1883) reported one case with bilateral optic atrophy. He believed the patient to have had symmetrical hemorrhages in the occipital lobes. Henschen's case (1890) revealed a gumma surrounding the optic chiasma. In the same year Von Graefe wrote of a case caused by a gumma extending from the base of the brain to the optic foramen. Lang and Beevor (1894) cited a case due to tabes dorsalis in which the nasal defect was attributed to selective optic atrophy. Rakowicz (1895) reported one case with optic atrophy of unknown origin. Bull also reported a case in that year with optic atrophy. Fridenburg (1896) cites an instance in which binasal hemianopia occurred 18 months after a man had been struck on his head. Burnett (1900) reported a case following a fall on the head. Eskridge, Schmidt and Wegner, and Veasey (1897) each reported a case with papilledema due to brain tumor. Coppez (1911) considered his case to be due to thrombi in both occipital lobes following cardio-renal disease. Cushing and Walker (1912) cited a case in Leber's disease. They also found that 17 out of 300 patients with intracranial neoplasm showed hemianopic defects with a tendency toward binasal blindness and all followed a far-advanced consecutive optic atrophy. They also presented one case due to pressure of sclerosed carotids. Heed and Price (1914) described one case due to tabes dorsalis. Hartman (1933) found his case to be due to post-hemorrhagic blindness. Banduin (1934) reported a case with acoustic neuroma of the cerebellopontine angle. Löwenstein (1935) reported a case with optic-nerve atrophy due to pressure of atheroma of the carotid arteries. Duncan (1936) presented two cases of intracranial tumor with optic atrophy (1) right cerebral hemisphere; (2) left frontal lobe; and one case of congenital lues with binasal hemianopia. Rouquier and Gaults's (1937) case was due to a probable arachnoiditis. Torkildsen's case (1938) was caused by a meningioma of the posterior cranial fossa.

Etiology

From the data presented in the literature, the following etiologic factors have been collected: (1) The most common cause is distension of the third ventricle by intracranial tumor with resultant pressure upon the optic nerve by the circle of Willis. (2) The optic atrophy itself may be a cause. This may be considered under: (a) tabes dorsalis, and quoting from Fuchs "We must assume that the tabetic degeneration, as a rule seated in the optic nerve may locate itself farther back in the chiasma;" (b) idiopathy; (c) Leber's disease; (d) trauma; (3) syphilis; (4) pressure on the lateral fibers of the chiasma by thickened arteriosclerotic internal carotids; (5) hysteria.

It might be noted that the lesions, ascribed to a central origin, were not studied *post mortem*.

Case report

R. F., a white boy, aged 13 years was first seen at the Children's Hospital (Chicago) on May 9, 1934, suffering from an acute right mastoiditis and a Streptococcus viridans septicemia. Ocular examination at this time was considered negative. A simple mastoidectomy was performed on May 19, 1934; the patient developed a lateral-sinus thrombosis on May 23, 1934.

The jugular vein was ligated and the clot removed. The postoperative course was extremely septic, with recovery two months from onset. A bilateral optic atrophy was discovered on June 18, 1935, while the patient was being treated in the endocrine clinic for obesity and a probable Lawrence-Moon-Biedl syndrome. X-ray report of skull on July 23, 1935, showed no evidence of brain disease but a slightly larger than normal sella. On August 7, 1935, a divergent

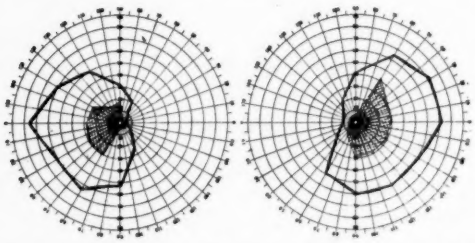

Fig. 1 (Gaynon). Visual field, showing the field for red cross-hatched.

strabismus was first noticed. All laboratory tests on spinal fluid and blood reacted negatively. Vision had been stationary for the past four years: O.D., 20/100; O.S., 20/200, with correction.

The father had died of nephritis at the age of 42 years; the mother had syphilis.

The patient was again examined on September 24, 1941, when the present defect was discovered. Physical examination revealed an intelligent, fairly obese youth of 13 years, whose only complaint was poor vision. Positive findings were mastoid scar, scar over right jugular vein, and tonsillectomy.

Ocular examination. Vision O.D., was 16/200, corrected to 20/200; O.S., 20/70+1, unimproved by correction.

Refraction. Vision O.D. with a +0.50D. sph. ⌯ 1.00D. cyl. ax. 90° was 20/100; O.S. with a +1.00D. sph. ⌯ 0.75D. cyl. ax. 90° was 20/70.

Both lids were normal. The conjunctiva was slightly injected, the anterior chamber of normal depth. The irides were blue, the pupils equal, reacting to light and accommodation and consensually. The optic discs were round, not elevated, pale white in color; their margins fairly distinct; the cribriform markings were not seen. The retinal vessels appeared to be normal. The macula seemed to be elevated. Tension (Schiötz) was O.D. 19 mm. Hg; O.S. 19 mm. Hg.

Blood tests showed: Wassermann reaction, negative; red blood cells, 5,440,000; white blood cells, 6,400; hemoglobin, 13.2 mg. Polymorphonuclear cells, 40; eosinophiles, 3; lymphocytes, 49; monocytes, 8.

On October 6, 1941, the spinal fluid was clear, the pressure 200 mm.; the cell count, negative; Pandy test, 0; protein, 13.6 mg.; the Wassermann reaction, negative.

GLUCOSE TOLERANCE TEST
100 mg. glucose given.

Fasting	*Blood*	*Urine*
	108	0
0.5 hr.	145	trace
1 hr.	135	0
2 hr.	139	0
3 hr.	135	0

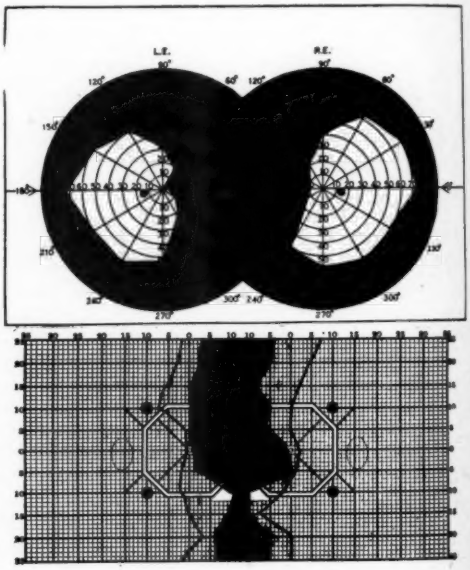

Fig. 2

The X-ray report, stated, as of October 4, 1941: "The stereoscopic X-ray examination of the skull of R. F. shows the sella to be shallow, but the clinoids and its floor are smooth. No abnormal calcifications are seen. The cranial bones and brain areas appear negative.

Ventriculogram reveals an atrophy at the chiasma. Hence, the lesion is due to a probable old healed arachnoiditis."

Visual fields are seen in figures 1 and 2.

SUMMARY

A brief review of the literature is given. An additional case is presented, due to a probable arachnoiditis at the chiasma.

ANTERIOR-CHAMBER IRRIGATION WITH SULFADIAZINE

JOSEPH LAVAL, M.D.
New York

The publication of the successful use of sulfanilamide in the anterior segment of the globe by Igersheimer (this Journal, 1943, v. 26, p. 1045) prompted me to report the following case.

Mrs. R. S., aged 60 years, a housewife, was admitted to the Mt. Sinai Hospital on September 10, 1943, for a cataract extraction following a preliminary iridectomy that had been performed in July. She was extremely myopic and mildly diabetic.

On the operating table an unusual complication arose. The episcleral-limbal suture which I use had been put in place, and the Graefe section had been performed, when suddenly the patient vomited. Before the speculum could be removed a good deal of the vomitus entered the cul-de-sac of the eye undergoing operation. The suture was drawn snugly and the lids were closed until the patient had freed herself of the gastric contents. Then the field was again prepared sterile and the eyelids opened. The vomited material was flushed from the cul-de-sac with saline solution.

It is considered that the gastric contents are sterile in the absence of achlorhydria but I was fearful of a possible infection even though I had good reason to know that the patient had a normal gastric acidity. Accordingly, a vial of 25-percent sodium sulfadiazine for intravenous use was opened and diluted with physiologic saline to 10-percent strength. A small 2-c.c. hypodermic syringe was filled with the 10-percent sodium sulfadiazine and a hypodermic needle was attached. The needle was inserted into the anterior chamber and the latter was irrigated. The irrigation was repeated twice and then the cul-de-sac was flushed thoroughly with more of the 10-percent sodium sulfadiazine. Following this the cataract extraction was performed without any further complications. The patient was given 15 grains of sulfadiazine by mouth five times daily for the first two postoperative days. Healing and convalescence were entirely uneventful. There was no undue reaction at any time.

Apparently the anterior segment of the globe can well tolerate 10-percent sodium sulfadiazine, and irrigation with this chemical is nonirritating. Whether it prevented an infection in this case is questionable, but the point is that the use of a 10-percent solution of sodium sulfadiazine is safe and entirely innocuous.

136 East Sixty-fourth Street.

EYES FROM AUTOPSIES*

SAMUEL GARTNER, M.D.,
VIRGINIA LUBKIN, M.D.
New York

We have been fortunate in obtaining permission for a great many autopsies

* From the ophthalmologic service of Montefiore Hospital.

which enabled us to remove the eyes. Most of them are removed through the conventional method of placing a speculum between the lids and enucleating the globe. If the autopsy includes the

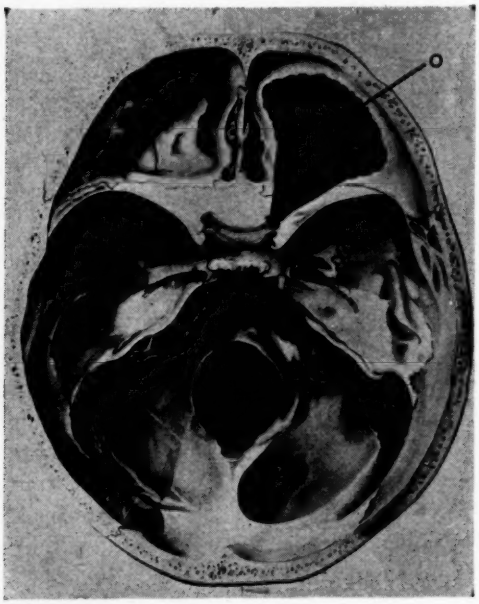

Fig. 1 (Gartner and Lubkin). Top view of base of skull. O, roof of orbit, which is removed.

brain, we have found it preferable to remove the eyes by the transcranial route, following the method Lambert[1] used. Figure 1 shows the roof of the orbit that is chiseled off, and through it the eye and the orbital contents are removed as one mass. The entire block of tissue is fixed and sectioned. Large slides are made that show the eye with a long section of optic nerve, the extraocular muscles *in situ*, and orbital vessels and nerves in their normal relationship, as in figure 2.

Great care must be taken to make the bodies presentable for burial, so glass eyes which match the original ones are placed in the sockets. Occasional embarrassment has been caused when the glass eye slipped out of position.

The following technique was developed to prevent such accidents. After enucleation, a wad of cotton, soaked in formalin, is inserted in the orbit to occupy the space of the globe. This hardens the tissues and forms a good support for the prosthesis. The glass eye is inserted and sutured into position so that it cannot move. The lids are closed, and then a large piece of cotton, saturated with formalin, is applied against the closed lids for a few minutes. The lids become quite rigid, so that they stay closed, and can be opened only to a small extent. A concealed intermarginal suture is occasionally used as an additional precaution.

In one method of attaching the sutures to the glass eye, holes were bored near the nasal and temporal ends with a dental drill. Once they were so prepared, they were satisfactory. However, we broke several eyes while drilling, and found the process tedious.

The method we now use is to apply the middle of a double-arm suture to the nasal and another to the temporal ends of the glass eye and fasten them in place

Fig. 2 (Gartner and Lubkin). Section of eye and orbital contents.

with Canada balsam. There are other adhesive substances which would do as well, provided they were not soluble in water and not affected by formalin.

A large curved needle is threaded on each end of the suture and carried through the tissues at the inner and outer canthus and tied inside the lids. This holds the eye securely and makes a very satisfactory appearance.

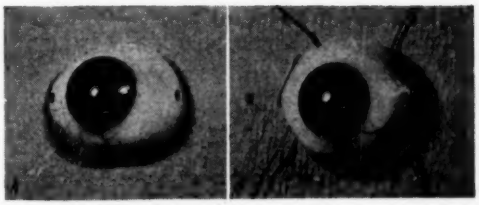

Fig. 3 (Gartner and Lubkin). Glass eyes prepared for autopsies. A, suture holes drilled in sides of eyes. B, sutures fastened to eye with Canada balsam.

Reference

[1]Lambert, R. K. Significant structural features of the ocular circulation. Jour. Amer. Med. Assoc., 1940, v. 114, p. 1802.

REFRACTION CLINIC*

Discussion by Dr. Albert E. Sloane†
Boston

A white woman, 55 years of age, became aware of poor vision in the right eye five months ago. She was told that she had retinal hemorrhages. Yesterday she was seen by another doctor who reported that both eyes show no evidence of pathologic change and advised that her refraction be tested. The patient complained primarily of ocular discomfort and blurred vision, especially when reading, following the onset of the "hemorrhages." Her present glasses are one year old and check to within 0.25D. of the findings as follows: She was wearing, right eye +4.25D. sph. ⌯ −.50D. cyl. ax. 90°; left eye +4.50D. sph. add +2.25D. sph.

Examination

Examination revealed: vision O.D. 20/200; with a +4.50D. sph. ⌯ −.50D. cyl. ax. 90° it was 20/50 "slow" (patient notes metamorphopsia). O.S. 20/200; with a +4.50D. sph. it was 20/20, add +2.50D. sph. for reading Well's no. 1. Pinhole disc over the right eye eliminated metamorphopsia but did not improve vision above 20/50.

Induced phoria. Vertical orthophoria. Distance 3Δ esophoria; near 2Δ exophoria. O.D. was the dominant eye.

During the examination this feature was noted: The vision was much better subjectively when the right eye was occluded, but immediately became uncomfortable and less clear when both eyes were open. Similarly, with the near glass, the patient had no difficulty reading Well's no. 1 type (fine Bible print), whereas with both eyes open the same type was barely legible and not comfortably so.

Discussion

The symptoms usually can be explained in two ways. First, a problem in refraction, or, secondly, a problem in binocularity. In the first instance refraction can immediately be eliminated as the cause of her symptoms since the findings do not essentially differ from the glasses worn. The problem of binocularity may be fur-

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†Director of Department of Refraction.

ther subdivided as (1) a disturbing heterophoria; (2) aniseikonia. Examination revealed 3D. of esophoria for distance, 2D. of exophoria at near, which is well within normal limits and very likely does not in any way contribute to the symptoms.

The problem of aniseikonia is not so easily dismissed. This could not be the usual type of aniseikonia because, if it were, the symptoms would be present for a long time. the refractive errors might be expected to vary in both eyes, and vision would more likely be somewhat similar in the two eyes.

The patient read 20/50 "slowly." This indicates that 20/50 vision was possible through studying the letters and probably also that 20/70, while legible, was probably read hesitatingly and "slowly." The fact that metamorphopsia was also present suggests an explanation of the acquired aniseikonia. Metamorphopsia, when present in regard to letters, usually indicates an irregularity in the plane of the retina in the macular area, so that one is getting a distorted image that is comparable to the distorting effect of an irregularity in the surface of a mirror. The pinhole, by obstructing some of this area and thus removing interference, tends to diminish the distortion of letters. But the fact that vision does not come up to normal indicates that there is not only an irregularity of the surface, but also, that there is some damage to the retina. In this way one understands that an aniseikonia of an irregular type has been produced which is, in turn, producing binocular discomfort. This is particularly distressing to the patient for two reasons. First, a well-developed fusion sense does allow easy suspension of vision in this eye, and, secondly, the affected eye is her dominant eye. Thus, she is more disturbed than if it were the nondominant eye that was affected. This may be compared to the embarrassment of a right-handed person who is suddenly required to use his left hand for precise work.

Therefore, the visual examination suggested some retinal pathologic change. It was found that the macular area showed an absence of the reflex and a peculiar marking of many irregular, small, yellowish areas which ordinarily are included under the general head of macular degeneration.

Diagnosis

Macular changes, retina, right eye, producing metamorphopsia with binocular discomfort.

Solution

All of these cases eventually are freed of symptoms through either of, or the combination of, two mechanisms: (1) the development of a compensatory suspenopsia in the affected eye, removing the interference to binocular vision from its distorted image; (2) a diminution in the amount of metamorphopsia as the pathologic process resolves.

Treatment

Explain to the patient the cause of his discomfort. He may occlude the affected eye when reading (usually with the hand), since it is usually during reading that symptoms are really distressing; for the metamorphopsia is disturbing in precise imaging but is not noticeable when looking at gross objects.

Questions

House Officer: How did you determine that the right eye was the dominant eye?

Dr. Sloane: There are many tests. The one I used in this case was to have the patient make a circle with the first finger and thumb of both hands. The person then sighted the muscle light through this

circle. Then, by noting which eye was looking through the circle, the dominant eye was determined.

H. O.: Would the 20/50 vision, O.D., without the metamorphopsia give interference to the O.S. with its 20/20 vision so as to produce symptoms?

Dr. S.: Yes, the difference in visual accuity in two eyes which have so recently seen equally will tend to produce symptoms, and the metamorphopsia seems to cause more disturbing symptoms particularly at near.

243 Charles Street.

SOCIETY PROCEEDINGS

EDITED BY DR. DONALD J. LYLE

COLLEGE OF PHYSICIANS OF PHILADELPHIA

SECTION ON OPHTHALMOLOGY

December 17, 1942

DR. ALFRED COWAN, *chairman*

EDWARD JACKSON, FOUNDER OF THE SECTION

DR. BURTON CHANCE delivered a memoir on this subject which was published in this Journal (January, 1943).

MEMOIR OF LUTHER C. PETER, M.D.

DR. EDMUND B. SPAETH presented this tribute, which will be published elsewhere.

MEMOIR OF WILLIAM T. SHOEMAKER, M.D.

DR. WILLIAM ZENTMAYER presented a paper on this subject which will be published elsewhere.

LIPAEMIA RETINALIS

DR. M. LUTHER KAUFFMAN presented two cases of lipaemia retinalis, both showing interesting and somewhat unusual findings. The first was not in a diabetic patient and was the third such case reported. This patient had suffered from chronic alcoholism for about two years, and also had had attacks of acute gastritis and vascular hypertension. In the second case there were marked changes in the walls of the retinal vessels and severe retinosis complicating the diabetes.

Neither patient was in coma on admission. If any acidosis was present in either patient it was not revealed by the carbon-dioxide combining power of the blood or by the urinalysis; pH of the blood was not determined.

The reason for lipemia becoming visible in the retinal blood vessels naturally depended to some extent on the lipoid content of the blood. Dr. Kaufmann suggested that another factor might be the size of the lipoid particles, which become larger in the presence of chemical or electrolytic imbalance. The increase in the size of the particles would make the lipoids more easily visible through the vessel walls. The particles in an emulsion tend to coalesce and become larger in anything but the optimum medium for that emulsion.

Discussion. Dr. Eunice L. Stockwell reported a case of lipaemia retinalis seen several months ago. The patient was a 41-year-old man who was admitted to the hospital with a typical history of diabetes mellitus of four months' duration. He had had no insulin before his admission. Dr. Stockwell saw him on the day of his admission. His blood sugar was 301 and the blood cholesterol was 1,456; his carbon-dioxide volume was 60

percent. The fundus in each eye showed the typical picture of lipaemia retinalis, the veins being full, cream-colored in appearance, and white in the terminal branches. The arteries were flat and ribbonlike, and approached the veins in size and color.

The patient was placed on insulin that day and the following day the typical picture of lipaemia retinalis disappeared. Although the veins and arteries were still slightly pale and the blood cholesterol was 1,401, the vessels returned to normal color.

Dr. Alexander G. Fewell congratulated Dr. Kauffman for picking up a case of lipaemia retinalis in a nondiabetic. He said he had seen only two cases of lipaemia retinalis. He thought the condition was much more common than reported. The case reported by Dr. Stockwell was a typical case of lipaemia retinalis, but the retinal picture entirely disappeared within 48 hours.

Very little had been added to Heyl's original ophthalmologic observation in 1886. With two exceptions, all previously reported cases had been in diabetics. One exception was the case reported by Wagner in 1922. The patient was a nine-year-old boy who had undergone radium treatment for leukemia. The other was that of a poorly nourished child, aged one year, with an associated xanthomatosis and enlargement of the spleen. In nearly all of the reported cases there had been an associated acidosis, and, in quite a number, xanthomatosis. The acidosis in these cases was usually severe and of short duration, but it might extend over a long period of time and be comparatively mild, as reported in McKee's last case. In only a few instances had there been a careful chemical analysis of the blood with studies of the various blood lipoids. In general terms, it might be stated that lipaemia retinalis appeared when the value of the blood fat rose above 3.5 percent and disappeared when it fell below 2.5 percent.

It had been suggested before that lipaemia retinalis was due to some condition other than the total amount of fat in the blood. This must be true, for a number of cases had been reported in which the fat content of the blood was high but no lipaemia retinalis was present. Before the discovery of insulin, practically all diabetic patients who developed lipaemia retinalis died in diabetic coma, but now with insulin these patients might live for many years.

Dr. I. S. Tassman asked Dr. Kauffman if he knew of any relationship between lipaemia retinalis and the white-cell count in these cases, especially since one case was reported by Wagner as occurring in leukemia. This brought up the question of whether the white count might have some relationship to the occurrence of lipaemia retinalis.

The other condition, such as xanthomatosis, was understandable because most of such cases were associated with diabetes; particularly those that occurred in young children in the form of the Schüller-Christian disease, which was a disease of the lipoid metabolism.

Dr. M. Luther Kauffman, in closing, thanked the discussers for their contributions. Concerning acidosis he pointed out that even though the urine and all other findings were normal, the chemists showed that it was impossible to say an acidosis did not exist unless pH determination of the blood was made. This was called an uncompensated acidosis.

As Dr. A. Fewell pointed out, these cases might be much more common than the literature indicated. The way to discover them was to examine ophthalmologically everybody admitted to the medical service of the hospital. The ratio of phospholipins to total fat was a point to be considered in causing lipemia to become visible in the retinal vessels. It might also have an effect on the electrolytic balance. The chemists pointed out

that any change in the electrolytic balance would have a tendency to make these fat particles coalesce.

His male patient had a leukocyte count below 9,000 at all times. The girl's count ranged from 8,000 to 16,000 but she had had frequent infections. He did not believe that the leukocyte count was a factor in lipemia.

An unusual case of birth injury of the eye

Dr. F. H. Adler and Dr. E. Gerard Smith presented the case of a 33-year-old physician who had sustained a birth injury, which was evident today by a large depression over the left frontal arc. He was refracted in 1931 and the findings were as follows: R.E. −2.50D. sph. ⊂⊃ −0.50D. cyl. ax. 60°, vision 20/20; L.E. −3.50D. sph. ⊂⊃ −2.25D. cyl. ax. 180°, vision 20/20. In 1935 the refractive error was R.E. −2.50D. sph. ⊂⊃ −0.75D. cyl. ax. 50°; L.E. −2.75D. sph. ⊂⊃ −2.75D. cyl. ax. 165°. The vision was 20/20 in each eye.

On reëxamination in 1938 the right eye was found to be about the same and the left had changed to −1.00D. sph. ⊂⊃ −2.75D. cyl. ax. 166°. Four years later the right eye showed little change but the left eye was now −3.25D. cyl. ax. 166°. When last examined in 1942 the right eye again showed very little change. The left eye was found to be +2.25D. sph. ⊂⊃ −3.50D. cyl. ax. 170°. This represented, over a period of 10 years, a total change of 5.75 diopters. The vision was still 20/20 in the left eye after each refraction.

Examination of the eyes showed a left hypertropia with fixation with the left eye. The right eye turned down and in, the patient suppressing the vision in the right eye. Due to facial asymmetry the left eye was 5 mm. higher than the right. The rotations of both eyes were full in all directions except on the left, where there was some limitation of outward movement. The right fissure was slightly wider than the left. The tension was 20 mm. Hg (Schiötz) in the right eye and 0 mm. Hg in the left eye.

Slitlamp examination of the right eye showed a few pigmented and nonpigmented deposits on the endothelium, but was otherwise negative. Examination of the left eye showed a marked chemosis of the conjunctiva of the whole lower cul-de-sac, limited to the lower bulbar conjunctiva. There were no signs of inflammation. There was a considerable amount of pigment on the anterior lens capsule, probably congenital in origin.

Ophthalmoscopic examination of the fundus of the right was negative. The fundus of the left eye showed slightly indefinite margins at the upper and lower poles of the disc. The retina showed some reflexes which were suggestive of retinal folds which may be due to an old retinal detachment.

Dr. Smith believed that the picture had a rational explanation. The globe might have been ruptured at birth. This rupture probably healed but may have left a fistula through which aqueous was escaping into the subconjunctiva and retroretinal spaces, which would account for the chemosis of the conjunctiva and possibly a forward displacement of the retina. The latter was suggested by the change in refraction.

Dr. F. H. Adler said he was sure that Dr. Smith, and he, and their very obliging patient, would welcome any suggestion that anyone might have that would differ from this explanation. He had not been able to find in the literature a case that duplicated this, and the only possible explanation that had occurred to them was what Dr. Smith had given. The patient's tension was zero with the tonometer. Dr. Adler had always taught his students, and had read in the literature, that a tension of approximately 25 mm. Hg (Schiötz) was necessary for the maintenance of the optical properties of

the eye. This eye had a visual acuity of 6/6 in spite of zero tension.

Discussion. Dr. James S. Shipman asked Dr. Smith if he had a record of what his patient's tension was at first refraction.

Dr. H. Maxwell Langdon asked if it was not remarkable that this case remained quiescent for a number of years. As he understood it, about 10 years ago the eye began to develop peculiarities and the patient was now 34 years old. What had happened which produced this peculiar series of changes in the course of events? Did anything happen that could have caused an opening up of a fistula? He was not suggesting that there was a fistula there, but he just wondered what caused the drainage to start. Could it have been that the pressure on the globe in taking the finger tension caused the leakage of aqueous which apparently was taking place?

Dr. Alfred Cowan thought it did seem remarkable that this conditon had progressed as described. Did not that seem unusual for an ordinary birth injury? In his experiences, birth injuries cleared up to a remarkable degree. It seemed from the appearance of the conditions which were considered to be due to birth injuries, that the eyes recovered to a much greater extent than if the same injury were sustained by an older person. So this case, if it really was a birth injury, was unusual in that it had become progressively worse.

Dr. Smith answered in regard to Dr. Shipman's question concerning what the tension was, that he got in touch with the men who examined the patient previously, and they told him his tension was normal when they examined him. With regard to Dr. Langdon's question about any injury, as far as he knew there was no subsequent injury. The patient did believe, however, that the chemosis below the cornea had always been present, to a greater or less degree.

PURTSCHER'S ANGIOPATHY TRAUMATICA RETINAE

DR. EDMUND B. SPAETH presented two cases of Purtscher's disease, one in abstract, the other in detail as to its clinical course.

The literature relative to this condition was discussed, for the etiology of the angiopathy is still not at all satisfactorily answered. This was reviewed in an attempt to determine, if possible, a consensus which might answer that question. The author himself suggested that fat embolism must be seriously considered as the possible cause of the retinal changes.

Discussion. Dr. James S. Shipman commented that this condition was not a recent discovery, having been described by Purtscher in 1910 in the case of a head injury. Even earlier than that, in 1900, Neck noted these fundus changes after compression or crush injuries of the chest. However, most of the literature on the subject was of European origin, and only in the past several years had it engaged the interest of American ophthalmologists. In a paper read before the American Ophthalmological Society and reported in the Archives of Ophthalmology in 1939, Bedell reviewed the literature; he had found 40 cases, and to these added 3 of his own.

In addition to the term "Purtscher's disease," the condition was also called "traumatic retinal angiopathy," "lymphorrhagia retinae," and most recently, "retinal teletraumatism." Knapp's "Medical ophthalmology," published in 1918, contained a description of the ophthalmoscopic picture, although it was given no definite name at that time. More modern textbooks included it under one of the above headings.

The usual history was a rather marked loss of vision in one or both eyes following a head injury, a compression injury of the chest or abdomen, or fractures of the vertebrae. The onset oc-

curred either immediately following the accident or the vision failed within a few days.

Externally there might be subconjunctival hemorrhages, often small, and ecchymosis of the eyelids and face as well as discoloration of the neck, chest, and upper extremities. There was no evidence of direct injury to the eye, such as laceration, nor was there any history of such injury.

Ophthalmoscopic examination revealed fairly typical fundus changes. Essentially, these were hemorrhages, exudates and edema, and dilatation of the retinal veins with constriction of the arteries. There might also be pallor of the disc, but more often this developed later when, in some cases, secondary optic atrophy ensued. A white appearance of the macular region had also been recorded as a late finding.

The hemorrhages might be small or massive, usually the latter. They might be flame-shaped or round. Most often they were found in the superficial layers of the retina, but a number of vitreous hemorrhages had been recorded. Stokes described a case in which there was such a massive hemorrhage in the macula that it had the appearance of "an island in a lake of blood." In addition, there was in *this case a round hemorrhage in the fovea.*

The edema and the exudates might be described together, these terms being ill defined by the various authors and in some instances, as in Bedell's paper, were used interchangeably. This was best shown by descriptions such as "the entire posterior pole was an immense milky white swelling covering the disc and retinal vessels" (Bedell); "a white, cloud-like plaque near the disc" (Bedell); "many discrete, elevated, whitish areas with round edges, some confluent, resembling cotton wool, and found along the superior and inferior temporal vessels" (Stokes). There might also be, as in the case presented by J. W. Smith, "a diffuse dense cloud or veil in the posterior vitreous" which entirely obscured the fundus and which was considered secondary to the retinal pathology.

The changes in the caliber of the blood vessels, that is, the constriction of the arteries and dilatation of the veins, was similar to that seen in thrombosis of the central retinal vein. It might be added here that the exudates were often found along the course of the vessels; hemorrhages, when small, were apt to be seen along the retinal veins.

The secondary optic atrophy was not a constant finding. A great many of the cases reported had been followed for only a short time. However, Bedell had the opportunity of seeing a case 15 years after he had made the diagnosis of traumatic retinal angiopathy. The disc in this instance was waxy pale, the vision markedly reduced as one would expect.

The mechanism of production of the hemorrhages and exudates was not entirely known. Purtscher's theory was that head injuries induced compression of the spine in its longitudinal axis, which, in turn, produced increased intracranial pressure. This pressure forced the spinal fluid through the intervaginal space into the nerve head and along the retinal vessels, extravasating into the retinal tissue through the ruptured perivascular lymph spaces. However, since the intervaginal space is not a true cavity, this was hard to accept. Friedenwald believed, as did Urbanek and Loewenstein, that the changes are due to fat emboli. If this was true, it would seem that more of these cases should be seen following fractures of the long bones. Experimentally, Willers trephined the skulls of rabbits and found that the brain could be made to bulge through the hole when pressure was applied to the thorax or abdomen. Each of these theories partially explained the pathologic physiology; no one of them completely explained it.

There had been only one case in which

histologic sections of the eye were made and microscopically examined. This was reported by Savitsky and Gross in December, 1935, before the New York Neurological Society. They found "round structures within the retinal layers, more in the superficial, which reacted to the stains for fat." There was a division of opinion as to the clinical diagnosis, several ophthalmologists considering retinitis albescens to be the true diagnosis rather than traumatic retinal angiopathy. For this reason it would seem unwise to accept the aforementioned pathologic picture as the usual one or as pathognomonic of the condition under discussion.

In none of the reports was any specific treatment suggested; treatment was not even mentioned. Presumably potassium iodide internally might be used to good effect.

The prognosis in these cases was variable. In a number of them there had been a return to normal vision, despite rather extensive retinal hemorrhages, exudates, and edema. However, secondary optic atrophy was always a threat in severe cases, and in others there had been reported permanent damage in the macular region consisting of fine dots and scattered pigment.

Dr. Edmund B. Spaeth said he had uncovered 51 relevant references, and these included some 70 cases. In regard to fat emboli, and the question which Dr. Shipman raised, was it possible that these were more probably the result of crushing injuries rather than otherwise? Injuries to the pelvis were not uncommonly followed by emboli and distant infarcts.

He did not believe that the histologic report mentioned was one of angiopathy traumatica retinae, and, with Dr. Shipman, he declined to accept that report as one for this condition.

Warren S. Reese,
Secretary.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

December 7, 1942

DR. ISADORE GIVNER, *presiding*

SYMPOSIUM ON TROPICAL OPHTHALMOLOGY

THE INCIDENCE OF OCULAR DISEASES IN THE TROPICS

DR. BERNARD KRONENBERG spoke of the ocular diseases in the tropics, and tropical diseases which have ocular symptoms. He stated that tropical ophthalmology is interesting because of the wealth of material resulting from poverty, neglect, ignorance, and lack of medical attention. Special climatic and dietary factors were discussed. Brilliant light, wind, and dust cause a large number of complaints.

Actually there are very few ocular diseases to be found only in the tropics. Among these are loa loa, microfilaria, and onchocerciasis. Not all diseases are found in all geographic areas of the tropics. Some are limited to the African or Caribbean regions, and others to the Asiatic regions. Trachoma and vernal catarrh, although found in some of the tropics are not seen in Puerto Rico.

The diseases which have ocular symptoms are divided into three groups: (1) Those found only in the tropics; (2) those found primarily in the tropics but also in other areas of the world; and (3) diseases which occur in all areas but which are rather frequent in the tropics.

Under the first group he mentioned yaws, trypanosomiasis, and yellow fever. In the second group he mentioned dengue, relapsing fever, Weil's disease, leprosy, malaria, and intestinal parasites. Malaria was discussed in detail and attention was drawn to the necessity of differentiating between amblyopia due to quinine and

malarial optic neuritis. In the third group of diseases he discussed cholera, dysentery, and typhus. Another group discussed was vitamin deficiencies.

Common ocular diseases of the colder climatic areas also occur in the tropics. He pointed out that uveal diseases and cataracts were more common in the tropics. Congenital cataracts were seen more frequently on smaller islands where there was much intermarriage. Refractive errors show nothing unusual except an early presbyopia.

Preoperative care must pay special attention to dietary deficiencies and the common occurrence of syphilis and poor dental conditions. The postoperative care, because of the heat and humity, must be a little different. Dressings must be changed more frequently.

In conclusion, he pointed out the definite existence of a field of tropical ophthalmology.

Ocular onchocerciasis

Dr. Edward B. Gresser said that onchocerciasis is an endemic roundworm infection found in widely separated tropic and subtropic foci, caused by the filaria of the genus Onchocerca, two species, Onchocerca volvulus and caecutiens, being parasitic for humans. The vector for human onchocerciasis is the biting black fly of the Sulidae family. The disease is of slow evolution, affects all ages, colors, sexes, and social stations, and is characterized by subcutaneous fibrous nodules or cysts located anywhere on the body and containing adult filaria. The mature filaria produce thousands of the markedly motile microfilaria which wander through superficial layers of the skin, accounting for the predisposition to ocular invasion with cysts above the neck line. Except when blindness occurs, the disease is neither debilitating nor disabling. The blindness can be of any grade or extent, unilateral or bilateral.

The microfilaria invades every tissue and layer of the orbital contents. The living parasite produces mild reactions, but when dead and disintegrating, the reactions are marked. The process becomes intermittent and chronic with acute exacerbations. Photophobia, lacrimation, and discomfort are constant. Pain is hardly ever marked, circumcorneal injection is invariable, and a rather torpid kerato-iridocyclitis is the rule, although a keratitis or iritis may be present alone. With the slitlamp, the living parasite can be discerned in the bulbar conjunctiva, cornea, and anterior chamber.

Pigment granules are spread over the surface of the iris, and, collecting in the lower angle, form, with inflammatory products from the iritis, a partial brown ring and a mass which contracts and causes pupillary deformity. Synechiae with pupillary occlusion and seclusion, secondary cataract, and occasionally secondary glaucoma occur. There may also appear chorioretinitis with degeneration of the pigmentary epithelium and clumping of pigment without relation to the vessels. The corneal changes are remarkable: A superficial punctate keratitis of grayish dots, a fraction of a millimeter in diameter, is found in the deeper epithelium and the outer stromal layers and usually in the lateral parts of the cornea. This is transient or permanent and in protracted cases occurs in the deeper layers. The corneal epithelium in later stages is irregular from cell swelling, desquamation, and scarring. Fairly common is a limbal vascularization accompanied by connective-tissue sheaths and inflammatory cells anterior to and often replacing Bowman's membrane. Descemet's membrane frequently shows folds or dehiscences.

Accompanying the cyst formation and

ocular disease is a disseminated dermatitis characterized by induration with a greenish hue. This discoloration, noted in the western world but not in Africa, may be due to difference in skin pigmentation.

No cure is known for onchocerciasis. Chemotherapy has not proved effective in any form. Hyperthermia, of theoretical value, has not been used. Most of the success achieved has been through surgical removal of the cysts and adult worms, presenting production of the microfilaria, and in preventing reinfection.

Discussion. Dr. Harry Most said that despite the great emphasis between the type of disease occurring in the western hemisphere and Africa, it is doubtful whether there are two valid species of Onchocerca. The African and Mexican disease should be considered the same, caused by the same parasite.

The black fly, which is the vector, lays its eggs in water, sometimes at the bottom of streams. Eradication of these flies, breeding in fast-moving mountain streams, is such a difficult problem that most people in preventive medicine consider it hopeless. It resolves itself into educating people not to go to the streams to bathe in the daytime, and to keep covered, since this is a day-biting fly.

Treatment is not so hopeless as Dr. Gresser pointed out. For the most part it is impractical to remove the nodules from all patients. It has been shown that it is possible to inject the nodules with bichloride of mercury, hexylresorcinol, arsenicals, or gentian violet.

Dr. Gresser, in conclusion, replied that while the literature is replete with therapeutic procedures and promises of apparent cures, in the last analysis, nothing touches the adult filaria, whether by injection of the cyst or by intravenous administration of drugs.

Experiences in an eye clinic in the Canal Zone

Dr. G. G. Marshall told of his experiences while chief of the eye, ear, nose, and throat section of the Gorgas Hospital, commencing in November, 1917. He found that natives of the tropics became presbyopic at an earlier age than those of the temperate zone, but there was no greater frequency of cataracts, partly explainable by the relative youth of most of the patients seen. Pterygia were very frequent among the Negroes, and bilateral pterygia were not uncommon. Simple conjunctivitis, due to the tropical sun and dust in the dry season, was prevalent but not serious. Koch-Weeks infection was more severe and corneal ulcers were frequent. The population of the city of Panama was said to be 30 percent syphilitic, and extensive choroiditis and optic atrophy were very common. Careful exclusion of emigrant laborers with eye disease made trachoma very rare.

Night blindness was prone to develop in the malaria-infected Negro living on a deficient diet. These patients recovered in 10 to 20 days when given fresh milk, vegetables, and cod-liver oil. No cases of malaria came to Dr. Marshall's clinic for treatment as such.

Leishmaniasis has been found in Brazil and Panama and has various manifestations. In Central America it is characterized by ulcers of the nasal mucous membrane, from which the conjunctiva is infected, and then the cornea, causing severe keratitis. Plasticlike deposits are found on the cornea and perforating ulcers may result. There is ciliary congestion, pain, and photophobia.

Dr. Marshall visited a leper colony at Palo Seco and treated all stages of the disease. Facial-nerve paralysis, ptosis, and loss of brows and lashes were early symptoms. The iris and cornea are the least resistant parts of the eye. On the

iris are pearllike bodies containing the bacillus which may extend to occlude the pupil. The iris finally atrophies. Corneal involvement may be punctate, annular, or diffuse parenchymatous and may progress to blindness. The vitreous, choroid, retina, and optic nerve are involved late. Cataracts are frequent but extraction is of little value as by this time other conditions make the eye hopeless.

Discussion. Dr. Connor said that in a case of malaria developing amblyopia he prefers quinine to atabrine. He has never seen a case of blindness in which the patient remained blind from quinine; and even though the drug would be continued in small doses until he recovered, eventually the vision would return.

Biomicroscopy in Trachoma

Dr. Milton Berliner said that the frequency of trachoma has so declined with the curtailment of immigration that the possibility of its presence may recede from the ophthalmologist's mind. While biomicroscopy has not made great contributions to the knowledge of trachoma, it has so correlated histologic changes with clinical findings as to permit early and differential diagnosis heretofore possible only by tissue examination.

Trachoma, like many other forms of chronic conjunctivitis, passes through the stages of hyperemia, neovascularization, papillary hypertrophy, and follicle formation. Neovascularization, following closely the hyperemia, is best seen in the tarsal conjunctiva at the beginning of inflammation or following prolonged mechanical irritation. Biomicroscopy shows small vascular tufts due to capillary proliferation from the normal vascular network just beneath the epithelium. The naked eye sees these as small red dots, easily mistaken for petechial hemorrhages, which may appear later. The organization of the capillary tufts results in the formation of papillae which can be seen in outline upon vital staining with azur II, even before they cause surface excresences. It is unknown whether some obscure chemotaxis or the firm attachment of the conjunctiva to the tarsus accounts for the appearance of these papillae in the tarsal rather than the bulbar conjunctiva. Follicles result from irritation causing lymphoid hyperplasia of the adenoidal layer of the tarsal conjunctiva. This reaction occurs in many viral, bacterial, and chemical conjunctivitides. Follicles and papillary hypertrophy may occur simultaneously and must be differentiated. The follicles are translucent protuberances between the papillae and are several times larger. There is a superficial vascular trellislike network in contrast to the central vascular tuft in the papillae. Ordinarily follicles shrink and disappear in time, but in trachoma they are invaded by a fibroplastic process, forming scars. Ordinarily follicles are found in the lower lid and fornix, but in trachoma they are chiefly in the upper lid.

At the onset of trachoma, when only mild congestion is seen with the naked eye, biomicroscopy may reveal typical features. Cuénod and Nataf have presented an excellent analysis of trachoma. Even when only a pinkish conjunctival blush is seen grossly, with high magnification and optic section are found small reddish dots just beneath the epithelium which are distinguishable as the newly formed perpendicular vessels of the incipient papillae. At the same time the small, round, slightly protuberant pale follicles are beginning to appear. An infiltrative haze at the limbus and proliferation of the superficial limbal arcades are seen in the early stages of trachoma.

The velvety appearance of the conjunctiva is the most striking feature of the second stage. Each papilla appears to be

a tile of mosaiclike pattern which is differentiated from the papillary hypertrophy of benign conjunctivitis by the combination of papillae, follicles, and corneal involvement. Follicles may appear on the caruncle and semilunar fold. High magnification after vital staining with azur II exhibits bluish follicles and papillae outlined by dark-blue dots, the whole resembling a hexagonal mosaic.

Bleblike formations resembling small "sago-grains" occur, especially on the upper tarsal conjunctiva and caruncle. These result from proliferative epithelial downgrowth into the submucosa with the formation of pseudoglands.

Stage three commences with delicate white lines of scar tissue between the papillae, and cicatricial invasion between the absorbing ruptured or necrotic follicles producing stellate scars first visible under the biomicroscope.

In the fourth stage the increased scar tissue results in a smooth, avascular white surface. A horizontal white band, the superficial line of Arlt, develops in the middle of the tarsus. Contraction of the scar tissue leads to the dire sequelae which are due mainly to deformity of the eyelid or dryness of the eye following destruction of the secretory elements. Further, the accompanying pannus leads to loss of vision.

Biomicroscopic study shows pannus to be an early specific part of the disease, not a complication. Almost immediately after the onset there is an alteration of the limbal relucence and vascularization which may be overlooked by ordinary methods of examination. Newly formed capillary loops extend in Bowman's zone. At this level many grayish points and lines produce a delicate haze. Within this area may appear small follicles, which, when, surrounded by a capillary network, form Herbert's rosettes. Follicles may be abortive or develop fully and then empty and organize, leaving small epithelialized depressions, usually near the limbus, known as Herbert's marginal pits. Irregular facets follow the breakdown of small subepithelial infiltrations. The capillary loops progress downward with a relatively horizontal border preceded by a grayish area of faint infiltration. At first the pannus is superficial, but in severe cases there is destruction of Bowman's membrane with deep scarring and vascularization and irregular thinning of the entire cornea. Secondary degenerative changes, such as calcareous deposits, fatty hyaline degeneration, and keratectasia are found with pannus of long duration.

Discussion. Dr. L. A. Julianelle emphasized the stages of hyperemia, neovascularization, follicle formation, and papillary hypertrophy as merely the result of a prolonged low-grade stimulus. Consequently the diagnosis of trachoma before pannus or scar formation is difficult.

The cause of trachoma is a virus, although it differs in incubation period, course, and immunity from the usual virus diseases. The inclusion body, believed by Dr. Julianelle to be the virus, can be seen in scrapings from the upper tarsal conjunctiva, stained with Wright's stain, and permits early diagnosis. Most viruses have high tissue selectivity and, regardless of their mode of entering the body, invade a particular tissue. Trachoma, showing the greatest selectivity, enters the eye only by way of the eye and can affect only the epithelial cells of the anterior segment. When clinical signs or symptoms appear a full-blown case of virus infection is already present. This leaves two possibilities for treatment: to prevent the virus from entering the cell, and to kill it while it is in the cell. The latter is the basis for treating trachoma, and is difficult as it calls for an agent to enter the cell and kill the virus but not the cell itself. Trachoma burns out spontane-

ously, and scar tissue, which is not susceptible to the virus, is formed in the process. Curettage, silver nitrate, and copper induce scar formation. The sulfonamides have had success ranging from zero in Dr. Julianelle's hands to 100 percent in the Indian Service. He is not convinced that they are effective in trachoma.

Dr. E. Waldstein described his own personal case of trachoma to illustrate the possibility of an acute onset with profuse discharge. He had seen favorable reaction to sulfonamide medication, but admitted its concurrent use with other forms of treatment.

Dr. M. Jaffe asked whether incision of the conjunctiva at the limbus, then stripping it back and curretting the blood vessels to the pannus, resulted in increased transparency.

Dr. A. Braley stated that the Indian Service has found that practically all of its cases recover in a short time with sulfonamide therapy. Periodic scrapings show disappearance of the inclusion bodies while the patient is under treatment. In a series of over 200 patients at Fort Apache, 10 showed no improvement and approximately 10 others recurred. Treatment of another group led to the conclusion that without adequate or sufficiently prolonged administration of sulfonamide, good results cannot be expected. He had never seen a proved case of trachoma failing to respond to sulfanilamide.

Dr. G. Epstein asked whether there can be trachoma without pannus.

Dr. Berliner, in closing, said he had not seen spectacular results with the sulfa group. He had not performed peritomy with curettage but had transplanted mucous membrane at the limbus and also inserted catgut subconjunctivally and found little reaction in the conjunctiva at the limbus; there was some clearing of the superficial scars but not the deeper ones. He said he has never seen trachoma without pannus.

Leon H. Ehrlich,
Secretary.

CHICAGO OPHTHALMOLOGICAL SOCIETY

December 21, 1942

Dr. Louis G. Hoffman, *president*

Scientific program

The structure and function of the retina

Dr. Stephen Polyak (by invitation) presented a paper on this subject.

Discussion. Dr. S. Gifford and Dr. H. J. Smith discussed this paper.

Kodachrome clinic

Dr. Robert Von der Heydt chose the following subjects for this presentation: 1. choroiditis; 2. rare clinical conditions; and 3. ocular trauma.

Clinical program

(Presented by the Department of Ophthalmology, University of Chicago)

Avascular interstitial keratitis—questionable

Dr. Louis Alkoff presented R. B., a white boy, who was under treatment for congenital syphilis. He was brought to the Out-Patient Department on May 6, 1942, because of clouding of the cornea of the right eye. On examination, the vision could not be tested, but he could see people and objects at a distance. A faint circumcorneal flush was present in the right eye. The cornea showed a dense, deep, wedge-shaped opacity, with the base from the 7- to the 11-o'clock position. The cornea of the left eye showed a fainter similar opacity. Slitlamp examination re-

vealed edema of the posterior stroma with roughening of the anterior and posterior epithelial surfaces. There were no vessels.

During the next two weeks the left eye became as clouded as the right and the patient began to stumble. He was admitted to the hospital and given 10 injections of typhoid vaccine. The opacities began to shrink and become fainter until they just covered the vertex of the cornea in each eye. No vessels were present; the opacity was still in the deep stroma with roughening of the anterior and posterior surfaces.

Retinitis proliferans, vitreous hemorrhage, rete mirabile of the right eye; peripheral retinal hemorrhage and exudate of the left eye

Dr Louis Alkoff said that C. K., a 42-year-old man, was first seen in October, 1942. The patient stated that two years ago he had suddenly seen black streaks before the right eye and his vision decreased immediately. He further stated that the vision in this eye had been 20/20 prior to that time. He was treated with O.T. injections for four weeks and the vision slowly improved. Since then he has had four similar episodes. There was also a history of sudden collapse of the right lung, for which he was treated with bed rest for nine months.

Vision, on admission, was R.E. 20/200, L.E. 20/13+1. The vitreous in the right eye showed many floaters, and the disc could hardly be seen. A dense white band of vascularized proliferating retinitis could be seen extending from the disc at the 2-o'clock position toward the temporal portion of the retina and vitreous. The macula was not seen. The fundus of the left eye was normal except for several small granular hemorrhages and irregular white exudative lesions, 6 disc diameters out in the periphery, at the 1-o'clock position. These were situated at the terminal branches of the superior temporal artery.

Laboratory examinations showed no findings of significance. The patient was to be tested for brucellosis, since several members of his family have had it, and it is endemic in his community.

Sympathetic ophthalmia

Dr. Maurice Drell said that J. O., a 64-year-old man who had been presented before the Society last year, had sustained an injury to the right eye in 1916, at which time a foreign body was removed. There had been recurrent pain and redness in the eye with general decrease in vision to perception of hand movements only at the time of the initial examination in July, 1941. Examination showed an iridocyclitis, complicated cataract, and an old scleral wound of the right eye. The left eye was clear.

Treatment consisted of the application of heat, atropine, and proteolac, but no marked change occurred until September 18, 1941, when he complained of pain in the left eye of five days' duration. The left eye showed an increased ray in the anterior chamber with cells, and engorged iris vessels. The left vision decreased from 20/20 to 20/50. The following day the symptoms had become more marked, and the pupil of the left eye had not dilated at all with atropine. The right eye was enucleated that day. On section this eye showed a typical sympathetic ophthalmia.

Since that time the vision of the left eye has improved to 20/16 but the patient has had a chronic iridocyclitis and a number of exacerbations and remissions. He has been treated with, and become sensitive to, atropine, scopolamine, homatropine, and duboisine. He has had salicylates, one course of neoarsphenamine, three courses of mapharsen, and the pupil has been kept dilated with 10-percent neosynephrin. The vision, at this time,

was corrected to 20/25+2 and he was able to carry on his work as a bartender.

ORBITAL NEOPLASM

DR. MAURICE DRELL said that J. D., a 44-year-old man, was first seen in 1935, when he had an otherwise asymptomatic exophthalmos of the right eye. Exophthalmometer readings were R.E. 23 mm., and L.E. 14 mm. There was some limitation of abduction and adduction of the right eye. The fundus and visual-field studies were negative. The chemistries, serologies, and urinalyses were negative; X-ray studies of the skull, orbits, optic foramina, and sinuses were also negative. The patient was seen by the Neurology Clinic, and a diagnosis of orbital tumor was made. Transfrontal exploration of the right orbit was performed but no lesion was found.

The patient returned in November, 1942, with an increase of protrusion of the right eye, and occasional redness after imbibition of alcohol. Exophthalmometer readings were R.E. 26 mm., and L.E. 15 mm. A mass was palpable below the superior nasal orbital margin; there was limitation of movement in all directions, especially superonasally; and some conjunctival congestion was present. Vision, with correction, was R.E. 20/200; L.E. 20/20. The fundus of the right eye showed some papilledema. The visual field studies showed enlargement of the blind spot and constriction of the lower temporal field of the right eye. X-ray studies showed what appeared to be a generalized decalcification of the posterior orbital wall, which was interpreted by the roentgenologist as secondary to the orbital neoplasm. An attempt was made at air injection of the orbit for further X-ray visualization, but upon insertion of the needle into the orbit, extravasation took place into the syringe, which suggested perhaps a vascular neoplasm. Further local procedures were postponed until the extravasation should have become completely absorbed.

DIABETIC RETINOPATHY; BILATERAL RETE MIRABILE

DR. NORMAN TEPPER said that L. W., a 52-year-old woman, came to the clinic four months ago. Four days previously she had noticed a sudden decrease in the acuity of her vision, everything beyond a distance of 20 feet appeared as a "gray fog." Lights seemed to have a red ring around them, and she saw dancing black spots in front of her eyes.

Vision, with correction, was R.E. 20/50−1; L.E. 6/200. Tension, taken with a Schiötz tonometer, was normal. External examination was negative. A bilateral veil of vascular network was seen extending into the vitreous from the disc to +12. Some small hard exudates and a few faint macular hemorrhages were seen. Examination of the urine showed specific gravity 1.032 and 4+ sugar reduction.

One week later, following homatropine-paredrine refraction, vision was R.E. 20/40−2; L.E. 20/200. At the present time, four months after onset, corrected vision was R.E. 2/200; L.E. ability to count fingers at 3 feet. Light projection and perception and central fixation were good. Red and green were named correctly. The vascular tree was not elicited in either eye. No fundus details were visible. The rete mirabile was present in each eye. In the vitreous of the right eye, at the 2-o'clock position, was a hemorrhage which showed early organization.

OLD EXTENSIVE OCULAR TRAUMA

DR. WILLIAM ROSENBERG presented M.P., a 52-year-old man, whose complaint in September, 1942, was loss of vision in the left eye of 25 years' duration, and fogging of the vision in the right eye of

5 years' duration. Vision, R.E., was 20/25 with +3.50D. sph. ⊂ +2.00D. cyl. ax. 85°; L.E. 10/200, with good fixation and projection; red and green were easily identified and the vascular tree was elicited in that eye.

Twenty-five years ago some iron chips had entered his left eye. Attempts to remove them with a giant electric magnet were unsuccessful. Enucleation was advised but was not performed. X-ray studies were not made. Since that time he had felt that the left eye was useless, although the vision had cleared. In the past five years he had been aware of spots in front of the right eye which moved with the eye.

Examination under paredrine homatropine cycloplegia improved the vision in the right eye to 20/16−4 with +3.25D. sph. ⊂ +2.75D. cyl. ax. 90°. Vision in the left eye was improved to 20/16−3 with +11.25D. sph. ⊂ +1.50D. cyl. ax. 95°. The fundus of the right eye was normal; slitlamp examination showed three superficial punctate opacities on the corneal vertex of the right eye. Examination of the left eye showed a corneal pathway with a rent in Descemet's membrane at the 10:30-o'clock position, 2 mm. from the limbus, leading to a hole in the iris at the 11-o'clock position. The anterior chamber was clear, though a few wisps of vitreous extruded into the chamber nasally. There was iridodonesis; no lens substance was visible although remnants of the posterior capsule were seen. There were several fine vitreous opacities. In the superior nasal quadrant, corresponding to the superior nasal vortex-vein emissaries, there was a large area of choroidal sclerosis. X-ray pictures of the orbit revealed no foreign body. It had apparently dissolved. The patient was fitted with a minifying lens (Catmin) and has fusion on the synoptoscope.

The diagnosis was traumatic corneal opacity and rupture of Descemet's membrane, hole in the iris, traumatic aphakia, traumatic superior nasal vortex-vein occlusion, and vitreous detachment, left eye.

CATARACTA COMPLICATA—ECTOPIA LENTIS

DR. WILLIAM ROSENBERG said that J. F. H., a 65-year-old man, gave a history of gradually decreasing vision for the past 15 years. He had been told that his vision was poor because of trauma, although he could recall only minor blows to his eyes. He was also told, seven years ago, that he had the beginning of a cataract in his right eye.

The vision in the right eye was 9/200; with the left eye he could count fingers at one foot. Light projection was quick and accurate and red and green were easily perceived. The vascular tree was elicited bilaterally. Examination showed marked bilateral iridodonesis with iris atrophy and bilateral dislocation of the lenses. Vitreous strands projected into the anterior chambers, and a foggy-white material was seen to surround the pupillary apertures. The pupils could be dilated only to 4 mm. The vision was improved R.E. to 20/50 with −4.50D. sph.; L.E. to 20/200 with −6.50D. sph. There was bilateral nuclear sclerosis. Many vitreous opacities were present in both eyes. The fundus of the left eye could not be seen. The fundus of the right eye, although somewhat atrophic, appeared to be otherwise normal. Visual field studies were normal. Medical and laboratory findings were essentially negative.

On June 28, 1942, a combined intracapsular loop extraction on the left eye was performed. The postoperative course was uneventful. Final manifest refraction improved the vision of the left eye to 20/30−3 with +9.75D. sph. ⊂ +3.50D. cyl. ax. 115°, and the patient was able to read newsprint rapidly with the addition of +3.00D. sph.

The diagnosis was bilateral subluxated

lenses, bilateral senile and complicated cataracts, and bilateral uveitis.

PERIVASCULITIS RETINAE

DR. BARBARA SPIRO presented E. S., a 31-year-old man, who in September, 1942, gave a history of the sudden appearance of a black streak in the upper visual field of his right eye, nine months previously. This cleared up in a day, and recurred six months later. He saw an eye physician who diagnosed an infected eye and treated him with injections. Five teeth were extracted to eliminate a possible focus of infection. There was no history of pain, redness, or tenderness of the eye.

The vision R.E. was 20/200, not improved under cycloplegic refraction; L.E. 20/13−4. The left eye was essentially normal. The vitreous of the right eye was hazy, containing a membranous opacity with dark-green nodules and black opacities. The vessels were thin and indistinct. A number of hemorrhages were seen around the macula and temporally in the periphery. A white mass, 1 P.D. in size, was observed temporal to the disc at the 9-o'clock position, with many small capillaries enmeshed in it. Similar masses were seen in the periphery. Slitlamp examination showed multiple golden deposits on the posterior surface of the cornea and in the anterior chamber and a few cells in the anterior vitreous.

Physical and laboratory findings showed no abnormalities. Two weeks later the vision in the right eye was 20/25 and the vitreous had cleared markedly. Four weeks later the vision was 20/16 and has remained so since. Vitreous floaters were still present and the fundus findings were the same except for the hazy vitreous.

The impression was that this patient had perivasculitis with proliferative retinitis. He was referred to the Neurologic Department, but no evidence of central-nervous-system involvement was found.

RETINAL DETACHMENT

DR. BARBARA SPIRO said that C. G., a 58-year-old woman, was first seen in April, 1942. She gave a history of having noticed flashes of light in her right eye in December, 1941. A diagnosis of detachment of the retina was made and she was treated with bed rest for three weeks. There was no history of injury to the eye.

Vision in the right eye was perception of hand movements at 4 feet, searching fixation, projection in all quadrants. Vision in the left eye was 20/13. The right eye was 10 degrees divergent. A total retinal detachment was found, extending to the disc; the highest point was seen with a +12.00 lens and the lowest point with a +8.00 lens. Old tears were present at the ora serrata and at the macula. The peripheral field of vision, taken with a flashlight, revealed a small area of vision in the upper nasal quadrant and one in the lower temporal quadrant.

Surgery was advised with poor prognosis because of long duration, completeness of the detachment, and the presence of the macular hole.

A microcoagulation detachment operation was performed on the right eye on April 27, 1942. The postoperative course was complicated by an acute upper respiratory infection. Three weeks later the retina was still elevated. Ten weeks later the retina appeared much flatter and the corrected vision was 20/200. The temporal and inferior operative reaction was more marked, with extensive pigment dispersion and white scarring. The peripheral field, taken with a 5/330 isopter, showed complete nasal constriction except for a 15-degree area surrounding the fixation point; there was about 25 degrees of temporal constriction.

Six months following operation, the corrected vision of the right eye was 20/50. The retina was fully attached, with a few faint folds in the macular area. There were some vitreous floaters,

but the patient complained only of diplopia.

IMPLANTATION CYST

DR. G. HENRY MUNDT said that this patient, a 66-year-old white man, came to the Clinic in November, 1938, and stated that a cataract extraction had been performed on the left eye seven years previously and on the right eye five years previously, in Italy.

Vision R.E. was 0.4; L.E. 1.0 (corrected). The following findings were noted: chronic trachoma, both eyes; bilateral aphakia; bilateral pterygium; iridectomy, left eye; and corneal nebulae. The tension was normal in each eye. Slitlamp examination showed proliferated epithelium and multiple yellow crystals in the posterior lens capsule of the left eye.

When the patient returned in March, 1941, a lesion filled with fluid was noted at the 9-o'clock position on the iris of the left eye, approximately 3 to 4 mm. in size. The pterygium was removed from the right eye. In May, 1941, he complained of tearing of the left eye. Fluorescein passed through the punctum and tear duct into the nose without difficulty.

He was observed at monthly intervals, receiving treatment for trachoma. In August, 1942, the corrected vision was R.E. 20/50, L.E. counting fingers at 6 feet. He still complained of tearing. Slitlamp examination showed epithelial proliferation over the surface of the posterior capsule, with several small cystic areas. The cyst on the iris had increased in size. A diagnosis of implantation cyst, in addition to a finding of Elschnig's pearls, was made, and X-ray therapy was advised.

Robert Von der Heydt.

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TREACHERY IN OPTICS

We are told that the fighting forces of the United States now number something like ten million. Generally speaking, these men represent perhaps the finest assembly of physical manhood that the world has ever seen. Many of them, unfortunately, will never return to their homes.

In combat, whether of land, sea, or sky, much depends upon the visual efficiency of this great body of fighters and auxiliaries. Some of them have been accepted for service in spite of the fact that their visual acuity without correction is a good deal short of normal, and that their efficiency must depend chiefly upon the wearing of correcting lenses. But in certain fighting services no significant degree of optical defect is tolerated. The requirements for officers of the Army and Navy are considerably higher than for the rank and file. The standards for the Navy and Marines are higher than those for the Army. Most exacting of all are the visual conditions laid down for the Air services.

As to every branch there is good reason for the visual standards prescribed. In all walks of life we are familiar with the handicaps created by imperfect ocular function. The schoolboy who reads badly

may do so either because of actual inability to see or because he has an eye fatigue which reduces his power of visual and mental concentration. Poor vision or eye fatigue in a workman is not infrequently responsible for injury or death to the man himself or to those around him. In like fashion, imperfect visual function may result in death and disaster among the armed forces.

Applicants for jobs with railroads or other industrial concerns often resort to special tricks in order to deceive the examiner. Sometimes they are successful, and by their scheming have added to the industrial hazard. The penalty paid by the individual, the organization, or the community may be serious.

The applicant for a special line of activity in the fighting forces may be shocked to find that he does not meet the visual requirements. It may be that he is genuinely desirous of giving the utmost possible service to his country in the emergency. Perhaps he would rather be an officer than a private, rather an air pilot than a ground worker. Frequently his first reaction is that the visual standard is unduly strict, that he is experiencing a personal injustice, and that the rule represents an obstacle which he should attempt to overcome. He forgets that such rules have usually been created out of the lessons of experience.

Somewhere around the recruiting station, or elsewhere, the disappointed applicant encounters someone who assures him that a certain "doctor" knows how to cure such optical imperfections. The candidate thinks the matter over and proceeds to visit the so-called "doctor," who, for anywhere from fifty to a couple of hundred dollars, undertakes to put the patient in a position to hurdle the service barrier. Usually, all the candidate gets for his money is disappointment and disgust, but now and then, either through devious methods or by the aid of certain mechanical "exercises" which stimulate a temporary forcing of accommodative or muscular adjustment, he succeeds in passing the test.

What happens to such a candidate? His inadequacy may be disclosed at some later stage of induction or training. Or he may have the good luck, especially if otherwise possessed of the best possible mental and physical equipment, to do valuable and efficient service. But he faces the risk that a visual breakdown, at some critical moment, will work mischief to himself and others.

It must be remembered that not only does bodily fatigue have a marked capacity to reduce ocular efficiency in the presence of a refractive or muscular defect, but eyestrain itself not infrequently causes general fatigue, with incidental reduction in central control. The hyperope, the astigmatic subject, even the myope who when in the pink of condition is able to force his visual apparatus to its peak of achievement, may find his efficiency seriously impaired in times of weariness or crisis. His hurdling of the visual barrier may then render him rather a liability than an asset to the armed forces.

Certain claims have been made as to ability to overcome, by training, the ordinary imperfections of red-green color vision. But careful research clearly indicates that any apparent success in this direction has been spurious and has depended upon acquiring, by infinite repetition, an extreme familiarity with the arrangement of a series of cards used in the testing of color vision. The individual who had gone through such a course of training would be just as likely to prove dangerous as any other victim of red-green color-blindness.

The man who has to undergo special training in order to cross the boundary line of visual requirement for a special

service would be wiser and more patriotic to give up the attempt to enter that service. If he maneuvers his way in, he may be a potential traitor to his country. Much more specifically may the accusation of treachery be applied to the pseudo-professional tricksters who fatten on this sort of enterprise.

W. H. Crisp.

EXERCISES TO IMPROVE VISION

Day by day the ophthalmologist is placed in an increasingly difficult position by his young patients and their parents who have heard tales of miraculous improvement of vision among their friends and acquaintances by eye exercises. Two groups are especially urgent, young men who desire to be enrolled in officers' training classes and whose vision is insufficient to meet the requirements of the armed branches in which they are interested, and, secondly, myopic girls who want good vision without glasses.

Most ophthalmologists for a long time "pooh-poohed" the idea that anything could possibly be done to help the visual acuity of these young myopic people except the prescribing of glasses. The fairly numerous cases in which visual training of one type or another has enabled a man to pass visual tests previously impossible for him are forcing the eye physician to give thought to the matter. The ever-recurring question is whether there is merit in this training that is being given. Is the visual acuity truly increased or is it the acquisition of accessory skills that gives success in those patients who do learn to read smaller letters on the vision chart? Do better psychologic approaches, the learning of tricks, or even the increased facility usually acquired by constant practice account for the results? The physician wants to know how frequently improvement can be obtained and how this is accomplished. Most doctors have neither the time nor the inclination to study the matter through for themselves, so they either tell their patients that they have no faith in these so-called exercises or that they must seek elsewhere if they want to try them.

In most large cities there are those who are giving these exercises. They tell the prospective patient that they may not be able to accomplish anything, thus putting the responsibility on the individual and furnishing themselves an easy excuse if success is not attained, and all at a very good price.

Undoubtedly ophthalmologists have turned a deaf ear to this physiopsychologic approach to the problem of sight. The concept is so different from that taught in standard textbooks on refraction, and the theory and methods of procedure have not even appeared in ophthalmologists' literature, that they are largely hearsay to most eye physicians. To them there seems something iconoclastic, almost heretical, in disregarding the dioptric system and considering the eye merely as a link in the path of the image to the brain, a link of practically limitless potentialities unless the eye is definitely pathologic.

Perhaps the ophthalmologist does not really want to believe that it is possible to improve vision materially by these methods. He has heard that they are enormously time-consuming, requiring from five to six hours of patient training daily for many weeks, and furthermore he fears that to learn the technique, supposing that it were truly worthwhile, would require much time and effort. Oculists have seen a marked turning toward orthoptics in recent years, and most of them have disliked giving the training. Then there has been the rise of the controversial subject of aniseikonia with its very special type of examination and unusual lenses. Eye

physicians may dislike to contemplate the possibility of another expansion of visual care.

The ophthalmologist now wants to know just what value there is in this training to improve vision. He is very, very skeptical and desires to move cautiously. How can this information be obtained? There seem to be two methods. First and most satisfactory would be carefully conducted clinical experiments. The set-up for this is complicated and expensive. It really necessitates the employment of technicians familiar with the methods of training generally used who must be paid, and the coöperation of ophthalmologists who are willing to give the necessary time to supervise, check, and criticize. Then, too, there must be willing and interested subjects for the experiment and an adequate set-up of suitable instruments, all very elaborate and costly; but, fortunately, this study is apparently soon to be undertaken in at least two places in this country, so that in a reasonable length of time the harassed ophthalmologist may expect some sort of an answer to his questions.

A second criterion on these so-called exercises may sometimes be made by the oculist on his own patients, if he can maintain friendly relations with those giving the training and will encourage his patients to return to him for systematic checks. The writer has been able to do this in a few cases, thus far without having found any material improvement in the subjects, but these are admittedly unfavorable patients with relatively high myopia and many of them have not followed out the prescribed treatment fully, especially not having given the necessary time to it. Most of those who claim to have had success with this training try to insist on almost full-time work over many weeks, whereas most patients want to continue concurrently other activities, such as school, and do the exercises in their spare moments only.

The type of pseudomyopia in which overefforts of accommodation, especially in reading, produce an associated convergence spasm, seems the only type that should reasonably yield to relaxation therapy, but this kind definitely should respond and it is probably on the favorable reaction of this fairly numerous group that most of the good reports rest.

Another point of view with regard to this visual training is presented in the first editorial in this issue, a viewpoint that certainly warrants careful consideration and is one more reason for attempting to arrive at some conclusion in the matter.

Lawrence T. Post.

BOOK NOTICE

AN INTRODUCTION TO CLINICAL PERIMETRY. By H. M. Traquair, M.D., F.R.C.S. 4th edition. Clothbound, 332 pages, 245 illustrations and 3 colored plates. London, H. Kimpton, 1942. Price $6.50.

This fourth edition of Traquair's highly regarded book, making its appearance in a war year, shows some change in its make-up, but little alteration in the text (see review of third edition, this Journal, 1938, v. 21, p. 1049). The book, as before, consists of 12 chapters. The early part of the book presents Traquair's classical concept of the nature and significance of the field of vision, forming the basis for the "quantitative" approach to perimetry. The second half of the book is a systematic description of the perimetric changes produced by disease of each part of the visual mechanism.

Several clinical entities receive a more thorough exposition in this new edition. Among these are a more careful elucida-

tion of the types of perimetric changes shown in Jensen's chorioretinitis juxtapapillaris; a description of the Foster-Kennedy syndrome; and a paragraph on optico-chiasmic arachnoiditis.

The portion of the Appendix dealing with the "Anatomical relations of the visual pathway" (section III; also sections IV and V) has always seemed, to this reviewer, to be of paramount importance in the appreciation of the perimetric changes for each part of the visual mechanism. The inclusion of eight new figures helps to focus the reader's attention on this section. They facilitate the organization of these anatomic relations into a mental picture which is necessary to the proper evaluation of the text. It is to be hoped that future editions will further emphasize this topographic-anatomic section.

This new edition is printed on thinner, less glossy paper, and there has been a reduction in over-all size, weight, and, best of all price.

Benjamin Milder.

OBITUARIES

AN APPRECIATION OF ALFRED VOGT 1879-1943

With the passing of Prof. Alfred Vogt, of Zürich, ophthalmology has suffered the loss of one who without a doubt has made the greatest contribution to ocular clinical diagnosis during the past quarter of a century.

His early studies of radiant energy and spectral analysis centered mainly on experimental work on the effects of ultraviolet and infrared light on ocular structures. The injurious effects of infrared light in industry and the possibility that with it cataracts could be made to order were disclosed. He showed the relative innocence of ultraviolet light, the supposed damaging effect of which, in his own words, had always been the "whipping-boy" in ophthalmologic literature.

Vogt's development of filters of copper sulphate and erioviridin B which, with an arc light as source of illumination, allowed of the examination of the fundus with red-free light, was a great advance in ocular diagnosis (1913).

By means of this illumination the yellow coloration of the macula could be observed in the living eye. There is an increased visibility of the nerve-fiber striation and in the reflection of retinal folds. The minute blood vessels could be followed practically into the macula. The progression of macular vacuole formation could be better observed. Macular pigment changes, especially the diagnosis of fresh pigment formation, which is yellow, may be differentiated from small black hemorrhages. In order to have a complete elimination of all red a microarc light had to be the initial source of illumination, as the filters excluded 97 percent of the light, leaving but 3 percent for observation.

The use of the older type of ophthalmoscope with mirror was necessary. Effort was made by manufacturers to circumvent these important necessities by building filters into the various types of electric ophthalmoscopes. This resulted in a semi-red-free illumination, thus defeating the real value of Vogt's method. Among Professor Vogt's publications are two monographs on red-free light.

His first "Atlas of slit lamp microscopy of the living eye" was published in four languages: German, English, French, and Italian (1921).

The second edition presented his life work, appearing in three large volumes (1930, 1932, 1942). The many thousands of colored illustrations were a monumental artistic achievement, not as yet equaled.

His book on the "Pathogenesis and operative therapy of retinal detachment" (1936) was a timely follow-up of the work of his fellow Swiss countryman Gonin.

During three decades ophthalmologic and medical journals continually carried a steady stream of Vogt's new contributions.

His investigation in genetic biology and on heredity culminated in the publication of an intensive study of senile changes in identical twins (Klinische Monatsblätter für Augenheilkunde, April, 1938). This contributed much of confirmative value to our knowledge of the many new evidences of ocular senility which he disclosed by the use of the slitlamp.

Among the many new uses of the latter was his utilization of the reflection of light on delimiting ocular surfaces. It allowed of a visibility of the individual endothelial cells on the posterior corneal surface. Here, and in this way only, individual living fixed cells may be seen.

Most important and of immense practical clinical value was Vogt's introduction of the very narrow slitlamp beam (1918-20), the utilization of an extremely thin optical cross-cut section. By means of this method the beam if well focused allowed of an accurate determination of depth within media. Thickenings as well as thinnings in certain areas of the cornea could be seen and compared. The topographic anatomy and histology of the lens, its nuclear zones and lamellae were accurately seen for the first time by means of this narrow beam. It also allowed of an exact determination of the ages of the many concentric nuclear zones.

Previous to these refinements slitlamp observation was limited to focal illumination and transillumination. These, however, disclosed such a wealth of new, heretofore unknown clinical pictures that many were astounded to observe this new world and were much enlightened. Hence the well-focused narrowed-beam refinement, with its increased possibilities, was usually overlooked, except by Vogt's disciples and followers. These, however, are obtaining a maximum of valuable clinical information.

A new era, bringing inestimable diagnostic possibilities as well as an interesting office diversion, has been the happy fate of Professor Vogt's followers. The use of the slitlamp at an expense of but little time has taught the art of accurate observation.

In 1939 his associates and friends from all over the world had published a Festschrift containing almost 100 articles to commemorate his sixtieth birthday.

He told me of his kidney involvement 20 years ago. In spite of this progressive affliction he had kept up a prodigious amount of clinical work, painstaking investigations, and had conducted a large private practice, drawn from all over the world.

So again, another ophthalmologic giant has passed on to immortality.

Robert von der Heydt.

SANFORD R. GIFFORD
1892-1944

American science in general and Chicago ophthalmology in particular suffered a grievous loss in the all too untimely death of Sanford R. Gifford on the twenty-fifth of February of this year. Up to eight days before, he had been feeling well, although extremely tired from the enormous overload of his University and private work. Four days later he entered the Passavant Hospital in a serious condition, and finally succumbed to pneumonia. In accordance with his expressed wish, a simple funeral service was held.

Sanford R. Gifford, born in Omaha on January 8, 1892, was the son of an

equally famous father, Harold Gifford, who left a great imprint upon American ophthalmology. His undergraduate collegiate work was completed at Cornell University, where he majored in literature. Later he received an M.A. degree from the University of Nebraska, and his M.D. degree from the same school in 1918. Following a short interneship in Wise Memorial Hospital in Omaha, he enlisted in the Army and was assigned as bacteriologist to Base Hospital 49 in Allery, France, with the University of Nebraska Unit, and later served with the Army of Occupation.

After his discharge from the Army, he went to the Nebraska Methodist Hospital, in Omaha, for a two-year interneship, and then entered practice with his father in that city, and at the same time was active in the University Ophthalmic Clinic. He spent the year of 1923 studying in various clinics and centers in Europe, including the Augenklinik in Freiburg, under Professor Axenfeld; at the Tübingen and Vienna clinics; with Morax in Paris; and at Moorfields in London. Publications of both clinical and investigative work began to flow from his pen, and soon the ophthalmologic profession became aware that there was another and greater Gifford on the horizon. In 1929 he was called to the Chair of Ophthalmology in Northwestern University Medical School, and shortly thereafter moved his residence to Chicago. Omaha's loss was Chicago's gain, for his ability and personality soon put him among the leaders of the profession.

There were four separate aspects to Dr. Gifford's professional life: his clinical work, his investigative work, his teaching, and his private practice; and I believe that subconsciously his interest rated them in that order. Although a research man of the first order, he was primarily a clinician, and as such his experience and judgment commanded national respect. His ophthalmic surgery was bold, and he pioneered in many fields of ocular therapy, but the safety and integrity of the eye under his knife was his first consideration. From a therapeutic standpoint, Sanford Gifford was undoubtedly the leader in this country, not only by his own work, but even more through his

Sanford Robinson Gifford, M.D.

outstanding "Handbook of ocular therapeutics." Even before his advent into medicine, he exhibited the mental curiosity of a true research man. He always wanted to know the why and wherefore of a problem, and that attitude led to some of his major investigative publications. The following are indicative of the scope of his work in this field:

Biochemistry of the lens. With Drs. J. E. Lebensohn and I. S. Puntenney: Archives of Ophthalmology, 1932, v. 8, p. 414; and American Journal of Ophthalmology, 1933, v. 16, p. 1050.

Visual sensation produced by roentgen and radium rays. With E. E. Barth: Archives of Ophthalmology, 1934, v. 11, p. 81.

Reaction of buffer solutions and of ophthalmic drugs. Archives of Ophthalmology, 1935, v. 13, p. 78.

Filtration experiments with the virus of inclusion blennorrhea. With E. B. Tilden: Archives of Ophthalmology, 1936, v. 16, p. 51.

Tendon transplantation for paralysis of the external rectus muscle. Archives of Ophthalmology, 1940, v. 24, November.

Central angiospastic retinopathy. Archives of Ophthalmology, 1939, v. 21, February.

Dr. Gifford was perhaps somewhat too impatient to be the ideal undergraduate teacher, but as a teacher of graduate students who approached his own mental level, he had no peer. Still his "Textbook of ophthalmology" for undergraduates is one of the outstanding manuals of its type; only those of May and Parsons are comparable. Under his inspired leadership the Department of Ophthalmology at Northwestern has done much to further the knowledge of this specialty among physicians in general practice. Furthermore, he had that rare gift of developing a "school" of young men, imbued with his ideals and following in his footsteps. Private practice was somewhat of a chore, although he enjoyed the human contacts involved, as well as the diagnostic problems associated with a large referred practice.

Sandy Gifford was the best "company" that a person could hope to have. Uniformly cheerful, he could always see the quirks in a situation, which he could usually top with an appropriate quip. He was very well read, especially in general science, and his memory was of the encyclopedic type. The French and German languages were second nature to him, and during the first Pan-American Congress of Ophthalmology, he delivered an impromptu speech in Spanish from the floor of the meeting. I well remember his earnest, albeit somewhat abortive attempts to master Arabic while we were attending the International Congress of Ophthalmology in Egypt, in 1937. Written English was quite a hobby, and he belonged to a literary society in Chicago, before which he delivered his famous essay on "Garlic and horseblankets," a masterpiece of analysis of the patients he saw in Omaha during his earlier days.

A very happy home life was his lot. Alice Carter Gifford maintained a smooth-functioning household and was the ideal helpmate for Sandy. The oldest son, Sanford, a graduate psychiatrist, is now in the Army, and the younger son, Carter, an undergraduate architectural student, is on duty with the Seabees.

We are going to miss Sandy more and more as time goes on. His influence on Chicago ophthalmology was great and was of that type that cannot be replaced easily. But for those of us who had the happiness of his friendship, there is a great void, an emptiness that his passing has left. Our sorrow is that of self-pity, for we think of Sandy and the joy that he gave us as The Immortal Bard has put it into the mouth of Claudius, King of Denmark,

"That we with wisest sorrow think on him
Together with remembrance of ourselves."

Harry S. Gradle.

ABSTRACT DEPARTMENT

EDITED BY DR. WILLIAM H. CRISP

Abstracts are classified under the divisions listed below, which broadly correspond to those formerly used in the Ophthalmic Year Book. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. General methods of diagnosis
2. Therapeutics and operations
3. Physiologic optics, refraction, and color vision
4. Ocular movements
5. Conjunctiva
6. Cornea and sclera
7. Uveal tract, sympathetic disease, and aqueous humor
8. Glaucoma and ocular tension
9. Crystalline lens
10. Retina and vitreous
11. Optic nerve and toxic amblyopias
12. Visual tracts and centers
13. Eyeball and orbit
14. Eyelids and lacrimal apparatus
15. Tumors
16. Injuries
17. Systemic diseases and parasites
18. Hygiene, sociology, education, and history
19. Anatomy, embryology, and comparative ophthalmology

3

PHYSIOLOGIC OPTICS, REFRACTION, AND COLOR VISION

Luckiesh, Matthew. **Test charts representing a variety of visual tasks.** Amer. Jour. Ophth., 1944, v. 27, March, pp. 270-275. (2 figures, 1 table, references.)

Messenger, H. K. **Principles of refraction.** Med. Clinics North America, 1942, v. 26, Sept., pp. 1595-1614.

The first half of this paper is a review of the basic principles of refraction, under the following headings: rays; object and image points, focus; the measurement of vergence; a simple means of calculating the distance of an image; the forms of an ophthalmic lens; refraction of the eye. The second half of the paper offers "a simple subjective method" for the correction of refractive errors, dealing with the determination of axis, cylinder, and sphere. (13 figures.) W. H. Crisp.

Pascal, J. I. **A rational basis for cross-cylinder tests.** Amer. Jour. Ophth., 1944, v. 27, March, pp. 280-281.

Pascal, J. I. **Simple transposition of obliquely crossed cylinders.** Dis. Eye, Ear, Nose, and Throat, 1942, v. 2, Nov., p. 349.

The author points out the practical usefulness of learning to transpose obliquely crossed cylinders to give insight into cross-cylinder work and cylindrical corrections generally. The mathematical method of accomplishing this transposition is explained.

Robert N. Shaffer.

Solandt, D. Y., and Best, C. H. **The Royal Canadian Navy color-vision test lantern.** Canadian Med. Assoc. Jour., 1943, v. 48, Jan., p. 18.

The construction of the Royal Canadian Navy color-vision-test lantern is described. It presents to the patient two white or colored lights which give a dimensional representation of the riding lights of a vessel with a beam of 25 feet at 2,000 yards. Various combinations can be used. By its use some 30 percent of those failing in the Ishihara test will pass. The authors think a large percentage of such patients have

strictly normal color vision, and that the others are anomalous trichromates with only slight defects. Test procedures are outlined and the evaluation of data obtained is discussed.

Robert N. Shaffer.

Sugar, H. S. **Binocular refraction with cross-cylinder technique.** Arch. of Ophth., 1944, v. 31, Jan., pp. 34-42.

Many persons who wear corrective lenses the prescription for which has been determined by accurate monocular refraction are uncomfortable during binocular vision. In many cases, says the author, the trouble is due to changes in the astigmatic axes when both eyes are used together for distant vision and during convergence accommodation. The paper analyzes such changes in a small series of 70 normal eyes with astigmatism of 1.00 D. or more, and explains the use of the cross cylinder to determine these changes under binocular fixation. All the patients had binocular single vision. Four explanations have been advanced; namely, lenticular changes, fusional compensation, torsional movements of the globe, and action of the extrinsic muscles in changing the curvature of the cornea.

The author has used the cross cylinder not only for binocular determination of the axis of cylinders for distance, but, more recently, for binocular determination of the axis of astigmatism for near vision when reading glasses alone were required. A table shows the change in axis from monocular to binocular testing as found in many cases.

As an additional conception in the attempt to get comfort with the binocular correction in difficult cases, the author takes up the subject of spherical equivalent. The spherical equivalent of a compound lens system corresponds to the dioptric midpoint of the interval of Sturm. Numerically, the spherical equivalent of a compound lens system in plus-plus or minus-minus combinations is equal to the spherical power of the system plus half the power of the cylinder. The spherical equivalent of a simple cylinder is equal to half the power of the cylinder.

A series of case reports is given showing the application of the spherical equivalent. The following combinations all have the same spherical equivalent: — 2.75 D. sph. + 2.00 D. cyl. axis 100°; — 2.50 D. sph. + 1.50 D. cyl. axis 100°; — 2.25 D. sph. + 1.00 D. cyl. axis 100°; — 2.00 D. sph. + 0.50 D. cyl. axis 100°.

In a given case these combinations were tried in the order listed. The most comfortable combination may be tried in a trial frame in the waiting room for a half hour as a test. In this particular case the third combination gave comfort and was prescribed. The visual acuity with this system was 20/25. (5 charts, references.)

R. W. Danielson.

4

OCULAR MOVEMENTS

Berens, C., and Romaine, H. H. **Postoperative cicatricial strabismus: results of the transplantation of Tenon's capsule.** Trans. Amer. Acad. Ophth. and Otolaryng., 1943, 47th mtg., Jan.-Feb., pp. 183-205.

This study was carried out in rabbits, using various techniques. The grafts used were free, pedicle, and apron; the substances, Tenon's capsule, amnioplastin, egg membrane, and peritoneum. Adhesions were produced by manipulation, scarification, and abrasion of the muscle with an instrument.

Pedicle grafts produced the best results. The apron graft apparently forms a tunnel in which the muscle acquires greater motility. Amnioplastin, egg membrane, and peritoneum were found unsatisfactory as substitutes for Tenon's capsule. Rough manipulation of the muscles or capsule produced adhesions, so that it is important to exercize the greatest care to avoid trauma to these structures. Reaction from suturing and from suture material should be avoided if possible. A suture material, nylon (6-0), has been used very successfully. In cases of complete fixation of the muscle to the sclera, transplantation of Tenon's capsule definitely improves motility.

Clinical reports of four typical cases are presented. Case 1 records a good result in postoperative cicatricial exotropia, case 2 a bad result in postoperative cicatricial exotropia, case 3 a good result in cicatricial exotropia following a second transplantation of Tenon's capsule in a patient whose cosmetic appearance and functional result had been worse after the first transplantation of Tenon's capsule, case 4 a good result in postoperative cicatricial esotropia. A table showing the findings in the 21 operations is included. (6 tables, 9 illustrations.)

Gertrude S. Hausmann.

Davis, W. T. **Differential diagnosis of paresis of the obliques and the superior and inferior recti muscles.** Trans. Amer. Acad. Ophth. and Otolaryng., 1943, 47th mtg., Jan.-Feb., pp. 206-209.

This paper was illustrated by a film, depicting the various types of paralysis of the ocular muscles. Until Bielschowsky formulated the differential diagnostic signs between palsy of the superior and inferior recti and the obliques, paresis of the vertically acting recti was frequently confused with that of the obliques. The outstanding differences are as follows: In palsy of the right superior oblique or of the left superior rectus, if the patient looks up and left there is a high amount of right hyperphoria, from overaction of the right inferior oblique. In palsy of the right superior oblique, if the head is tilted toward the right shoulder, there is little or no vertical deviation. In palsy of the right superior rectus no change in vertical deviation occurs on tilting the head toward either the right or the left shoulder.

Gertrude S. Hausmann.

Foster, J., Pemberton, E. C., and Freedman, S. S. **Graded squint operations.** Trans. Ophth. Soc. United Kingdom, 1942, v. 62, pp. 320-329.

The authors tabulate the results obtained with the number of millimeters of recession or resection and the degree of squint corrected. The operations were carried out under general anesthesia up to 14 years of age, and under local anesthesia above that age.

One-mm. recession of the internal rectus produced an average of 2.9° correction. One recession in four produced a surprise result, and squints which were variable prior to operation remained variable after operation. The unintended vertical displacement obtained in a simple recession or advancement could not be controlled by raising or lowering the insertion of a horizontal muscle. (3 tables, 1 diagram, references.)

Beulah Cushman.

Guibor, G. P. **Recession of the inferior-oblique muscle from the external-rectus approach.** Amer. Jour. Ophth., 1944, v. 27, March, pp. 254-257. (13 illustrations, references.)

Harris, Wilfred. **Ataxic nystagmus. A pathognomonic sign in disseminated sclerosis.** Brit. Jour. Ophth., 1944, v. 28, Jan., pp. 40-42.

The author reports a condition he calls ataxic nystagmus. The eyes turned laterally and the conjugate action appeared weak. The right eye, when looking toward the left side, did not reach the inner canthus. At the same time the left eye showed a coarse nystagmus, as though outward movement could not be maintained because of weakness. Usually the nystagmus of the left eye continued as long as the patient looked to the left, the right eye remaining stationary in its position short of the inner canthus. It was this discrepancy between the movements of the two eyes which suggested the term "ataxic nystagmus." Three case reports are given. Beulah Cushman.

Lynn, Beatrice. **The treatment of concomitant and latent squint.** Trans. Ophth. Soc. United Kingdom, 1942, v. 62, pp. 344-351.

The author outlines the results obtained five years after the inauguration of an orthoptic department in two hospitals. The steps in the treatment were estimation of the error of refraction, occlusion to prevent or overcome amblyopia exanopsia, establishment of simultaneous macular perception, increasing power of fusion, and correction of deviation (by operation, if necessary), together with version and vergence exercises and stereoscopic vision.

Among the stimulating exercises advised for suppression is frequent attendance of the cinema or teaching by cine-projectors, because the amblyopic eye sees moving objects more easily than stationary ones. Observations seem to demonstrate that if a child has a squint before the age of two years it is unlikely that fusion will be established, but it is of vital importance to prevent amblyopia and to increase the lateral movement of each eye. Suppression must be overcome before a strong sense of fusion can be established. When this has been done, operative procedures may be undertaken and exercises resumed. (One table, 3 diagrams.) Beulah Cushman.

Spaeth, E. B. **Congenital blepharoptosis—a classification. The principles of surgical correction.** Trans. Amer. Acad. Ophth. and Otolaryng., 1943, 47th mtg., March-April, pp. 285-300.

The importance of early diagnosis and treatment of congenital ptosis is stressed. Of the cases here discussed, 37 percent were complicated by such symptoms as muscle imbalance and torticollis, loss of vision either single or binocular, curvature of the spine, and spasm of the occipitofrontalis. The cases are divided into nine groups: (1) unilateral ptosis without involvement of the superior rectus muscle, (2) unilateral ptosis with involvement of the homolateral superior rectus muscle, (3) bilateral ptosis without involvement of the superior rectus muscle, (4) bilateral ptosis with involvement of the superior rectus muscle, (5) unilateral ptosis with weakness of both superior recti muscles more marked in the homolateral eye, (6) ptosis with more or less complete third-nerve and even sixth-nerve paralysis, (7) ptosis with the classical jaw-winking reflex, (8) ptosis with Duane's retraction syndrome, (9) ptosis with neurofibromatosis. The three main principles to be considered in surgery for correction of ptosis, all of them necessary at various times in these cases, are: use of the occipitofrontalis, use of the superior

rectus muscle (if normal) and shortening of the levator. General ptoses, classified under 1, 2, 4, 5, and very occasionally 9, are best corrected by levator advancement with usually partial tarsectomy. Ptosis classified under 3 is the ideal indication for the Motais-Parinaud technique. For ptosis classified under 6, early extraocular muscle surgery is important. It should be completed before the ptosis itself is corrected. For ptosis classified under 7 and 8 a complete tenotomy with tenectomy of the levator should be followed several weeks later by a Reese orbicularis transplant to the occipitofrontalis. Ptosis classified under 9 should usually be corrected by a Hess procedure. (21 illustrations.)

Gertrude S. Hausmann.

Wheeler, J. R. **A review of the treatment of concomitant and latent strabismus.** Trans. Ophth. Soc. United Kingdom, 1942, v. 62, pp. 353-356.

Binocular vision, a late acquisition in phylogeny, can only be acquired postnatally. Binocular coördination probably develops between three and six months of age, but may not be stable for a number of months. From 18 months to two years development proceeds rapidly, and by six years of age binocular coördination is firmly established. Rarely does so-called concomitant strabismus commence after that age. The later the squint develops the better the prognosis. The author states that his aim in treating concomitant strabismus is to obtain two eyes cosmetically straight, and with good binocular vision. As causes of concomitant squint he cites physical defects, fusion defects, refractive errors, and sometimes a psychologic factor.

Beulah Cushman.

5

CONJUNCTIVA

Perkins, J. E., Korns, R. F., and Westphal, R. S. **Epidemiology of epidemic keratoconjunctivitis.** Amer. Jour. Pub. Health, 1943, v. 33, Oct., pp. 1187-1198.

During five months beginning September, 1942, an extensive epidemic of keratoconjunctivitis was studied in a Schenectady factory. For military reasons, the names and the number of cases involved are not given. Approximately 4 percent of the entire personnel were affected, and of these 56.1 percent showed the disease in both eyes and 85.5 percent developed corneal opacities. In only 19 percent had the corneal opacities disappeared at the end of two months. The peak of the epidemic was reached five weeks after onset, and after this the epidemic subsided gradually until the thirteenth week, when a slight increase again occurred. The groups most affected were first physicians and nurses, and next, employees exposed to ocular foreign bodies. A higher rate was also noted in firemen who slept in dormitories. Secondary cases in the households of those affected were approximately 4.9 percent, most frequent in adult females and milder in children. The cause is probably a virus transmitted from person to person (respiratory?). Only a small percentage of those exposed, however, develop clinical symptoms. The virulence of the virus, the amount of exposure, and the resistance of the host are apparently the factors which determine who will be infected and how severely. A rigid aseptic technique was employed, consisting of washing hands routinely between patients, using a separate sterilized dropper for each pa-

tient, and frequent sterilization of instruments and solutions.

Charles A. Bahn.

Terrizzano, M. F., and Terrizzano, A. J. M. **Tuberculous keratoconjunctivitis treated with roentgen therapy.** La Semana Med., 1944, v. 51, Jan. 6, pp. 22-23.

The patient was a girl of 13 years. The disturbance involved the cornea and the conjunctiva of the right upper eyelid. Biopsy from the conjunctiva showed numerous tuberculous follicles. The interstitial keratitis was sufficiently dense to prevent a view of the iris, and the vision was reduced to light perception. Enucleation was recommended but refused. Four roentgen irradiations were given in the course of a month, each of 200 r. One similar treatment was given the next month, two in the third month, two in the fourth month, and one each in the fifth and sixth months of treatment. Eleven months after the treatment was initiated the vision of this eye had returned to normal, although there were two small corneal leucomas.

W. H. Crisp.

Wold, K. C. **Epidemic keratoconjunctivitis.** Minnesota Med., 1944, v. 27, Jan., p. 25.

Epidemic keratoconjunctivitis is to our knowledge a virus disease characterized by an acute conjunctivitis, lid edema, lack of purulent discharge, preauricular adenopathy, and in some cases the appearance of corneal maculas which occasionally reduce visual acuity for a period of several months. Treatment is mainly symptomatic, although the use of convalescent serum or early small doses of X ray have in some cases seemed to influence the otherwise self-limited course of the disease.

Owen C. Dickson.

6

CORNEA AND SCLERA

Lloyd, R. I. **Less evident causes of lowered acuity in senility.** Amer. Jour. Ophth., 1944, v. 27, March, pp. 232-243; also Trans. Amer. Ophth. Soc., 1943, v. 41. (13 illustrations, references.)

Perkins, J. E., Korns, R. F., and Westphal, R. S. **Epidemiology of epidemic keratoconjunctivitis.** Amer. Jour. Pub. Health, 1943, v. 33, Oct., pp. 1187-1198. (See Section 5, Conjunctiva.)

Robson, J. M. **Experimental corneal ulcers.** Brit. Jour Ophth., 1944, v. 28, Jan., pp. 15-25.

A résumé of the production of corneal ulcers and the application of treatment at the different stages of ulcer formation is given. The ulcers were developed by injection of B. pyocyaneus. pneumococcus, staphylococcus aureus, hemolytic streptococcus, and the human and bovine strains of the tubercle bacillus.

The work was started in 1940 for the purpose of investigating experimental mustard-gas lesions on the eye and their treatment. It was found that secondary infection played an important part in determining the severity of the lesions. Local treatment with chemotherapeutic agents decreased the severity of the primary reaction and also the tendency of the rabbit eyes to develop delayed vascularized keratitis. The infections produced by different types of organisms could be influenced by chemotherapeutic agents and antiseptics. The methods of administration of such drugs are compared. (2 illustrations, references.)

Beulah Cushman.

Terrizzano, M. F., and Terrizzano, A. J. M. **Tuberculous keratoconjuncti-**

vitis treated with roentgen therapy. La Semana Med., 1944, v. 51, Jan. 6, pp. 22-23. (See Section 5, Conjunctiva.)

Wold, K. C. **Epidemic keratoconjunctivitis.** Minnesota Med., 1944, v. 27, Jan., p. 25. (See Section 5, Conjunctiva.)

7

UVEAL TRACT, SYMPATHETIC DISEASE AND AQUEOUS HUMOR

Leathart, P. W. **Parasympathetic iridoplegia.** Trans. Ophth. Soc. United Kingdom, 1942, v. 62, pp. 306-308.

The author uses the term parasympathetic iridoplegia to describe a condition which he has found at rare intervals following purulent sinusitis. The pupil on the side of the sinusitis is widely dilated and will not contract to light or convergence; and the near vision is blurred. The extrinsic ocular muscles and optic nerve are unaffected. The condition is exactly opposite to that found in Horner's syndrome. The lesion is located in the region of the ethmoid sinus, and involves the constrictor and convergence reflex arcs after the afferent and efferent limbs have left the third-nerve nucleus. Recovery has occurred in a few weeks without other treatment than eserine, and leaving no disability. (One diagram.) Beulah Cushman.

Lowenstein, O., and Levine, A. S. **5. Pupillographic studies (periodic sympathetic spasm and relaxation, and role of sympathetic nervous system in pupillary innervation).** Arch. of Ophth., 1944, v. 31, Jan., pp. 74-94.

This paper presents detailed pupillographic study of a woman patient aged 22 years, with a syndrome of periodic sympathetic spasm and subsequent relaxation. Part of the time the eyes were under normal conditions and at other times they were under the effects of various drugs. The patient suffered from a progressive degenerative condition of the central nervous system, characterized by gradual development of motor and sensory disturbances, peculiar gait and periodic spastic extension at the wrist and flexion of the ulnar three fingers, which preceded by some seconds the periodic pupillary phenomena.

Impulses traveling over the sympathetic pathways produce pupillary dilatation consisting of several dilatation phases at an average speed of 0.4 mm. per second, followed by contraction at an average speed of about 0.07 mm. per second. The primary interval in the present case was 2 mm.

The light reflex of the pupil, usually considered parasympathetic, contains in its phase of contraction some factors, which are determined by the functional state of the sympathetic system, whereas the primary phase of contraction depends predominantly on the parasympathetic system. When the secondary and tertiary phases in contraction are weak, cocaine will improve them.

When the pupil is contracted with physostigmine and the primary phase of contraction is poor, it can be improved by sympathetic spasm resulting from neutralization of physostigmine by epinephrine. When the pupil becomes small because of poor sympathetic innervation, hypersensitivity to cocaine and hyposensitivity to physostigmine develop. Conversely, when the pupil becomes large under the influence of hyperinnervation by the sympathetic system, hypersensitivity to physostigmine and hyposensitivity to cocaine develop. (12 figures, references.)

R. W. Danielson.

Ray, B. S., Hinsey, J. C., and Geohegan, W. A. **Observations on the distribution of the sympathetic nerves to the pupil and upper extremity as determined by stimulation of the anterior roots in man.** Annals of Surg., 1943, v. 118, Oct., p. 647.

Knowledge of the preganglionic sympathetic outflow to the upper extremity and pupil of man has been incomplete because of deductions from indirect methods and experimental observations of laboratory animals. In a series of 18 patients in whom the cervicothoracic region of the spinal cord was exposed and the anterior nerve roots were electrically stimulated, the following observations were made: In ten patients changes in the pupil showed that sympathetic innervation traveled through one or more roots between levels of cervical 8 and thoracic 4. In 16 patients in whom preganglionic innervation to the hand was investigated it was found that there was variation in the upper and lower levels within limits of the first and tenth thoracic. After division of all anterior roots containing sympathetic fibers to the hand there was a return of sympathetic activity within ten weeks. This may indicate a functional reorganization through other pathways. The observations emphasize the necessity for recognizing the variability in the levels of sympathetic control to the upper extremity and pupil in man.

Theodore M. Shapira.

Reese, A. B. **Pigment freckles of the iris (benign melanomas); their significance in relation to malignant melanoma of the uvea.** Amer. Jour. Ophth., 1944, v. 27, March, pp. 217-226; also Trans. Amer. Ophth. Soc., 1943, v. 41. (14 illustrations, 5 in color, references.)

8

GLAUCOMA AND OCULAR TENSION

Evans, P. J. **A note on iridencleisis.** Brit. Jour. Ophth., 1943, v. 27, Dec., pp. 548-550.

The author describes his modification of the operation of iridencleisis. He states that it is indicated in cases of noncongestive glaucoma, either chronic or subacute. If the iris is still elastic and the pupil responsive to eserine, the author makes a peripheral iridectomy by an oblique incision including only about two thirds of the withdrawn iris, but sufficiently long to provide a tongue which will reach the subconjunctival pouch through the lips of the incision. The temporal pillar is reposited into the anterior chamber, while the nasal pillar is included in the lips of the incision. The final result retains the sphincter action of the pupil and provides an adequate fistula. Gentle massage and 0.5-percent pilocarpine are used for one to three months following the operative procedure. (2 illustrations.) Beulah Cushman.

Gibson, G. G. **Transcleral lacrimal-canaliculus transplants.** Amer. Jour. Ophth., 1944, v. 27, March, pp. 258-269. (5 illustrations.)

9

CRYSTALLINE LENS

Dunlap, L. G. **Safety in cataract extraction.** Trans. Pacific Coast Oto-Ophth. Soc., 1942, v. 27, pp. 150-155. (See Amer. Jour. Ophth., 1943, v. 26, Nov., p. 1242.)

Foster, J. **Preoperative cultures and antisepsis.** Trans. Ophth. Soc. United Kingdom, 1942, v. 62, pp. 329-335. (See Section 2, Therapeutics and operations.)

Samuels, B. **Pathologic changes in the lens associated with nontraumatic iritis.** Arch. of Ophth., 1944, v. 31, Jan., pp. 8-17; also Trans. Amer. Ophth. Soc., 1943, v. 41.

The author had described in one previous article the structural changes in a lens during the active stage of a corneal ulcer; and, in a second paper, the changes that may result long after an ulcer has been replaced by scar tissue. The present paper deals with the microscopic study of lesions occurring in a lens in nontraumatic iritis. Ordinarily a lens remains clear after a first attack of iritis. It is only after repeated attacks that opacities appear in the lens. In this study, microscopic examination was made of the lenses from twenty globes with nontraumatic iritis. A majority of the globes had been enucleated because of tuberculosis of the uvea, usually of the conglomerate type. In two globes cysticercus was discovered; one had been involved in sympathetic ophthalmia, and in one a retinoblastoma was present. In one globe the condition was mistakenly diagnosed as retinoblastoma, and two specimens showed syphilitic iritis.

In six cases in which iritis had been severe there was evidence of coincident glaucoma. In fourteen cases the capsule showed ruffling and folds. In many specimens it was noted that the disintegration of lens fibers seemed to have antedated proliferation of the epithelial cells. (8 figures, references.)

R. W. Danielson.

10

RETINA AND VITREOUS

Agatston, S. A. **Clinical analysis of fifty cases of diabetic retinitis.** Med. Record, 1943, v. 156, Aug., p. 482; Sept., p. 553.

The author surveys fifty cases of diabetic retinitis. The duration and severity of diabetes are not definitely related to the retinitis, nor does complete control of diabetes arrest the retinitis. Absence of changes in retinal veins and arteries is a favorable prognostic sign. The author thinks the first sign of retinitis is not lipoid exudate but petechial capillary hemorrhage. Venular and capillary pathology offers the most logical explanation of fundus pictures, because the advanced retinal changes are so frequently out of proportion to existing arterial or renal disease. The presence of engorged retinal veins indicates a tendency to thrombosis with danger of large hemorrhages. Such changes render an eye unfavorable for intraocular surgery, because of the probability of postoperative hemorrhage.

Robert N. Shaffer.

Berens, Conrad. **An illuminated retractor for eye operations, especially for detachment of the retina.** Amer. Jour. Ophth., 1944, v. 27, March, p. 281. (One figure.)

Black, G. W. **Detachment of the retina.** Trans. Ophth. Soc. United Kingdom, 1942, v. 62, p. 311.

The author reports a case of bilateral detachment of the retina treated by the Guist operation upon the right eye in 1933, and in which in 1940 extracapsular lens extraction was performed on the same eye with final visual acuity of 6/24 with +5.00 sphere.

Beulah Cushman.

Dickson, W. E. C., Pritchard, G. C., Savin, L. H., and Sorsby, A. **Two cases of visible emboli in retinal arteries.** Brit. Jour. Ophth., 1944, v. 28, Jan., pp. 1-14.

The author gives two cases of visible

obstruction of retinal arteries. For one case histologic illustrations were obtained. In one case there was a heart lesion with vegetations, complicated by embolic processes, in the absence of arteriosclerotic changes or thrombus formation. (19 illustrations.)

Beulah Cushman.

Eisaman, J. L. **Chronic occlusive arterial disease (arteriosclerosis obliterans), associated with retinitis pigmentosa.** Amer. Heart Jour., 1943, v. 26, Dec., p. 836.

A 56-year-old male gave a history of chronic occlusive arterial disease affecting particularly the lower extremities. Associated with this condition there was a typical picture of retinitis pigmentosa with no apparent familial background. This case suggests that pathologic changes may not be localized in the retina but may occur as a part of a similar disorder elsewhere in the body. Owen C. Dickson.

Gifford, S. R. **Proliferating retinopathy in diabetes.** Quarterly Bull. Northwestern Univ. Med. School, 1943, v. 17, Winter Quarter, p. 252.

The contrast between ordinary diabetic retinopathy and proliferating diabetic retinopathy is pointed out. The author thinks the larger retinal veins are involved in the latter condition and that thrombosis of these veins may be the precipitating factor. The only case the author has seen from its onset was initiated by vitreous hemorrhages, followed by the usual blood-vessel and connective-tissue invasion of the vitreous.

In four cases out of six studied, increased capillary fragility was found. Cases with proliferating retinopathy are more likely to develop gangrene, and vice versa, showing that both have changes in the peripheral vessels. Gifford points out the similarity between this condition and retinal periphlebitis. It is suggested that in all such cases careful study of the peripheral circulation be made, including pressor tests, heparin clearance, capillary fragility, and estimation of blood ascorbic-acid. The author concludes: "It is conceivable that if such tests were employed in every serious case of diabetes, the development of this tragic ocular condition might be prevented."

Robert N. Shaffer.

Gifford, S. R., Bauman, L., Jacobi, H. G., and Berens, C. **Symposium on diabetic retinitis.** Amer. Jour. Digestive Dis., 1943, v. 10, Sept., pp. 329-339.

Gifford: In the largest group of retinal retinopathies the basic picture is of small round deep hemorrhages with small white discrete deposits in and about the central area. Frequently superimposed are fine flame-shaped hemorrhages in the nerve-fiber layer, usually due to associated vascular sclerosis. A second group of retinopathies is characterized by large, often preretinal, hemorrhages, usually followed by a variable new-vessel and connective-tissue formation. A third group presents retinal venous thrombosis.

Diabetic retinopathies are more frequent in older patients, and are not proportionate to the severity of the diabetes but to its duration. Ascorbic acid and vitamin-B complex do not improve the ocular condition because they do not cure the capillary fragility. Citrin, when available, should be tried.

Bauman: Hypertension is an aggravating factor in the production of diabetic retinopathies. Retinopathies are very frequently accompanied by gangrene and other foot complications of diabetes. Both result from sclerosis of the peripheral arteries with interference

of circulation, and both are largely preventable. Lack of restraint and coöperation is the principal cause of poor control in diabetes. Protamine zinc insulin alone or combined with ordinary insulin controls most patients. A new product, globin insulin, which apparently has the advantages of both, will soon be on the market.

Jacobi: Glycosuria and polyuria are harmful because they disturb the electrolyte balance. Too rigid diet restriction may be just as harmful as excessive laxity. A mild glycosuria may not be harmful in the elderly or arteriosclerotic diabetic, being a safety measure against insulin shock. Retinal lipemia and similar lesions are much less frequent with a low-fat régime.

Berens: No single ophthalmoscopic sign is pathognomonic of diabetes, although perimacular and other retinal changes are suggestive. Hypertension is apparently not the principal cause of retinal hemorrhages in diabetics. Bacteria and metabolic toxins may be factors. In patients with increased capillary fragility no improvement has followed the use of vitamin-B complex and ascorbic acid. Charles A. Bahn.

Leopold, I. H. **Diabetic retinopathy.** Arch. of Ophth., 1944, v. 31, Jan., pp. 96-101.

This review of the literature discusses the etiology of diabetic retinopathy under the headings of hyperglycemia, vascular status, vitamin therapy, endocrine glands, and chemical constituents of the blood, especially fat.

Diabetic retinopathy has been variously attributed to uncontrolled hyperglycemia prior to administration of insulin (with the suggestion that the retinopathy is essentially a capillary disease).

Other special fields of study, here reviewed, include: dilatation of the retinal veins with production of punctate hemorrhages; experimental work as to the role of the endocrine glands in diabetes; and fat metabolism in relation to therapy. (Bibliography.)

R. D. Danielson.

Lewis, E. M. **Angiomatosis retinae** (successful treatment of one case). Trans. Amer. Acad. Ophth. and Otolaryng., 1943, 47th mtg., May-June, pp. 354-363. (See Amer. Jour. Ophth., 1943, v. 26, Dec., p. 1347.)

Lijo Pavia, J. **Detachment; reattachment; tears and cyst of the retina.** Rev. Oto-Neuro. Oft., 1942, v. 17, Oct., pp. 107-117. (See Amer. Jour. Ophth., 1943, v. 26, June, p. 644.)

Newton, F. H., and Schade, A. H. **An experiment to determine the conservation of vitamin A in the eye under strain.** Ohio State Med. Jour., 1943, v. 39, Sept., p. 827.

The authors assume that night blindness is associated with vitamin-A deficiency and that soft tones on the walls of some industrial plants reduce eye fatigue. Six female employees whose work required very sustained and accurate fixation under artificial light were dark-adapted for two minutes, then subjected to a powerful light for two minutes, and then placed in the dark again, with a view to testing regeneration of the visual purple. Those who worked under a Verd-A ray lamp claimed to feel less fatigued than those who worked under a frosted lamp. In two cases with excessive eye fatigue, two hundred thousand units of vitamin A were given for a week. Less fatigue was then experienced, and the eyes felt more comfortable with a Verd-A lamp than with a frosted lamp. (Graphs.)

Charles A. Bahn.

Sal Lence, J. **Specular pulsatile zones of the vitreous.** Arch. de la Soc. Oft. Hisp. Amer., 1942, v. 1, Oct., pp. 336-338.

With the aid of the Gullstrand ophthalmoscope, and examination of the eyes of more than 1,450 school children, the author has been able to observe brilliant reflexes in the fundus, especially along the veins. These reflexes are caused, according to the author, by internal reflection of the beam of light in the framework of the vitreous. Contrasted with the pulse of the retinal arteries, which is only present in certain pathologic conditions, the pulse of the reflexes described is a constant finding, especially in the eyes of young people; and its absence would indicate a pathological condition.

Ramón Castroviejo.

Wahrsinger, P. B. **Severe pre-eclampsia with separation of the retina.** Amer. Jour. Obstet. and Gynecology, 1943, v. 46, Oct., p. 371.

Appearance of retinal separation in severe pre-eclampsia is extremely infrequent but has been noted on several occasions. The author presents the following case: A 33-year-old woman, in her second pregnancy, was seen during the fourth month of gestation. All examinations were normal. The previous pregnancy had ended in spontaneous abortion. During the eighth month the patient was examined at home. She gave a history of nausea and vomiting of 24 hours duration, and an episode of temporary blindness. There was marked edema of face, conjunctivas, hands, legs, and ankles. The blood pressure was 195/110 mm. Upon hospital admission, urinalysis showed hyaline and granular casts. Sedatives, glucose, Karell diet, and Strognoff treatment were used. Ophthalmoscopic examination showed: right eye large retinal detachment in nasal half of fundus; small detachment below the optic nerve; left eye, large retinal detachment above and a small detachment below the optic nerve. The retinas and the margins of the optic nerves of both eyes were edematous, the margins of the discs blurred, the arteries narrowed. Retinal detachments and edema of the retina were probably due to subretinal and intraretinal transudates. The second day after Cesarean section, the edema of the retina subsided, but the detachments were of the same size. On the twelfth day post partum, the vision cleared and the blood pressure was 150/80 mm. Six weeks post partum, there was complete recovery from the retinal detachments. All findings were normal, the blood pressure 120/80 mm. The diagnosis was: severe pre-eclampsia with the retinal separation as a complication. Theodore M. Shapira.

11

OPTIC NERVE AND TOXIC AMBLYOPIAS

Agatston, S. A. **Congenital cyst of the optic nerve.** Amer. Jour. Ophth., 1944, v. 27, March, pp. 278-279. (2 illustrations, 1 in color, references.)

Anderson, W. A. **Medullated nerve fibers.** Trans. Ophth. Soc. United Kingdom, 1942, v. 62, p. 343.

The fundi of a patient seen in 1943 at the age of 60 years, with normal vision, showed medullated fibers completely covering the edge of each disc. In September and October, 1940, the same patient presented himself with pain on movement of the left eye, reduced vision, optic neuritis, and some macular changes. The vision of the right eye remained normal. After re-

covery there was optic atrophy of the left eye, without light perception, and all of the medullated fibers had disappeared. The medullated fibers of the right eye remained as before.

Beulah Cushman.

Gottlieb, B. **Retrobulbar and peripheral neuritis.** Rev. Oto-Neuro-Oft., 1942, v. 17, Sept., p. 183.

An alcoholic and chronic pipe-smoker exhibited classical symptoms of retrobulbar neuritis and peripheral neuritis of the upper and lower extremities. There was an associated achlorhydria. During treatment the patient was allowed to smoke, but alcohol was limited. He received orally high doses of vitamin-B complex and parenteral vitamin B_1. After five weeks his vision was markedly improved, as was also the peripheral neuritis. Edward Saskin.

King, A. B. **Tunnel vision.** Quarterly Jour. Studies on Alcohol, 1943, v. 4, Dec., p. 362.

The term "tunnel vision" has been used to describe an effect of alcohol on the eyes. In the experiments of the author no tendency to blot out the sensitivity of the retina to visual stimuli at the periphery was found in five subjects who ingested 30 to 75 c.c. of absolute alcohol. The author concludes that "tunnel vision" is an inappropriate figure of speech. The reaction time of the eye in accommodating was found to be slowed. Robert N. Shaffer.

McGregor, I. S. **A study of the histopathological changes in the retina and late changes in the visual field in acute methyl alcohol poisoning.** Brit. Jour. Ophth., 1943, v. 27, Dec., pp. 523-543.

The author reviews the literature concerning the ocular and general findings in methyl-alcohol poisoning, then describes four acute cases. The retina was examined after routine staining with hematoxylin and eosin in bulk. No changes in the retinal ganglion cells could be attributed definitely to the acute poisoning by methyl alcohol, either in the form of lipoid changes in the ganglion cells, in the size and shape of the ganglion cells, or in nuclear content. The optic nerves in each case appeared normal.

Four patients who had been blind in the acute stage were examined a year later. One had no defect of vision, the second a bilateral scotoma, the third an absolute scotoma in one eye while the other eye was normal, and the last a relative scotoma in one eye with good vision in that eye and the other eye normal.

The author concludes that histologic examinations of the retina so soon after poisoning are rare, and that the evidences in acute poisoning are not sufficient to show whether it is the retinal tissue or the nerve tissue which primarily succumbs. The follow-up suggests that the poison acts on the center of the optic nerve. (7 illustrations, references.)

Beulah Cushman.

Mata López and Barthe Pastrana. **A new case of Cruzon's disease.** Arch. de la Soc. Oft. Hisp.-Amer., 1942, v. 1, Oct., pp. 329-335.

The authors report a new case of Cruzon's disease with the typical characteristics of oxycephaly, bilateral exophthalmos, divergent strabismus, diminution of vision, and facial deformities. The pathogenesis of this disease is discussed. In cases accompanied by progressive optic atrophy and diminution of vision, the authors advocate freeing the optic nerve at the optic foramen by the operation of Hildebran,

who has performed it with success in five cases. Ramón Castroviejo.

12

VISUAL TRACTS AND CENTERS

Evans, J. N., and Browder, J. **A problem of split macula.** Arch. of Ophth., 1944, v. 31, Jan., pp. 43-53; also Trans. Amer. Ophth. Soc., 1943, v. 41.

The course and function of various fasciculi of the optic pathway have held the interest of ophthalmologists and neuro-anatomists for many generations, because knowledge in this field has a broad application to the diagnosis and treatment of ocular and cerebral disease. There are two general avenues of approach for studies of these fiber bundles. One may produce experimental lesions in various laboratory animals and study the resulting progress of fiber degeneration, or one may make functional studies on human beings who are suffering from certain localized lesions. Functional studies made on human subjects can be supported by anatomic evidence only when the injury or disease is observed at autopsy or operation. A review of the literature disclosed but two instances in which the optic chiasm had been split anteroposteriorly.

The major therapeutic problem that presents itself in attempted removal of a craniopharygioma is minimal injury to the hypothalamic nuclei and their connecting pathways. To this end three methods of surgical approach were utilized: first, removal of the tumor piecemeal, the optic apparatus being left intact; second, section of the one optic nerve in order to obtain a better exposure of that part of the tumor situated beneath the chiasm; third, splitting of the optic chiasm and division of the anterior communicating artery.

The authors report in detail the case of a 13-year-old boy with such a lesion. At operation the chiasm was divided with precision. The fields taken later showed classic bitemporal hemianopsia with a fingerlike absolute central scotoma which protruded from the blind field above the point fixed. The central scotoma could not cover the point fixed, since, macular function being impaired, the paramacular retina was used for this purpose. The true position of the macula was necessarily represented by some point within the fingerlike scotoma.

If the concept that the macular fibers form a semidecussation in the chiasm is adopted, then in the case cited the surgical procedure divided all the crossed macular fibers, with the result that the corresponding part of each macula was rendered functionless. This scotoma may then be assumed to represent the approximate size and shape of the part of the macula supplied by the crossed fibers.

The problem of fixation was of course encountered in the taking of the fields. A great many methods have been suggested for maintenance of fixation, but in this study the authors utilized the principle of the position and size of the normal blind spot and the adjacent angioscotoma. The use of these landmarks insures accurate fixation in normal subjects to within 0.25 degree. The authors conclude that the visual acuity resulting from division of the crossed macular fibers is approximately 20/40, which permits the subject to read and write such material as he is apt to need in average daily life. It is argued that the entire macula must be supplied by both crossed and uncrossed fibers. (References, 5 figures.)

R. W. Danielson.

Krug, E. F., and Echlin, F. A. **Tuberous sclerosis.** Arch. of Ophth., 1944, v. 31, Jan., pp. 68-73; also Trans. Amer. Ophth. Soc., 1943, v. 41.

A case of tuberous sclerosis is reported in detail in a young boy who was also afflicted with adenoma sebaceum. The cerebral tumor was removed by surgery with some loss of mental and physical powers. The visual acuity and visual fields were impaired and partial optic atrophy appeared. The term tuberous sclerosis was first used by Bourneville to describe a rare form of cerebral sclerosis occurring chiefly in children and young persons. Vogt, in 1908, established the clinical syndrome of mental deficiency, epilepsy, and adenoma sebaceum. Adenoma sebaceum, or nevus sebaceus, a disease of congenital origin, is characterized by pinhead-sized, yellow or reddish, waxy papules, situated on the flush areas of the face.

Tuberous sclerosis is so named because of the potato-like appearance of the morbid growth. Neoplasms of this type may appear in the kidney, the heart, the lungs, the spleen, and the retina. Signs of intracranial tension are evident in early adult life, as epileptiform attacks. In the absence of such manifestations, a facial eruption should arrest the attention and suggest the presence of tuberous sclerosis, and concurrent papilledema at once suggests intracranial neoplasm. Associated retinal tumors are comparatively rare. (3 illustrations, references.)

R. W. Danielson.

Mello, A. R. de. **Hereditary degeneration.** Arquivos Brasileiros de Med., 1943, v. 33, nos. 3-4 and 7-8, 90 pp. (Received in reprint.)

This ninety-page article reports in detail 16 cases studied in the neurologic clinic of Austregesilo, at Rio de Janeiro. The literature is reviewed. No general conclusions are presented. Most of the patients belonged to familial groups, one family including six affected offspring. Some details of the eye condition are given in most of the cases, the predominating ocular feature being atrophy of the optic nerve, sometimes accompanied by other fundus lesions and sometimes by disturbances of the ocular muscles. (51 illustrations, separate bibliographies of the Brazilian and extra-Brazilian literature.)

W. H. Crisp.

Michaelson, I. C. **Ocular manifestations of neuroses found among soldiers.** Brit. Med. Jour., 1943, Oct. 30, p. 538. (See Section 17, Systemic diseases and parasites.)

Stewart, S. G., Randall, G. C., and Riesenman, F. R. **Hysterical homonymous hemianopsia with hemiplegia and hemianesthesia.** War Med., 1943, v. 4, Dec., p. 606.

A soldier suddenly developed partial left hemiplegia with homonymous "macula-sparing" hemianopsia, consistent with a diagnosis of an organic lesion of the brain on the right side. The clinical course, together with the objective neurologic observations, indicated a hysterical condition. Psychotherapy produced prompt and dramatic cure.

Robert N. Shaffer.

13

EYEBALL AND ORBIT

Abbott, L. D., Mandeville, F. B., and Rein. W. J. **Complete roentgen and ophthalmologic examination for ochronosis in two alcaptonuric children.** Virginia Med. Monthly, 1943, v. 70, Dec., p. 615.

Two Negro children, aged 10 and 15

years, with alcaptonuria showed no positive roentgen evidence of joint change, ligament calcification, or calcium deposits in the ear cartilages. Ochronotic pigmentation of the scleras and conjunctivas was not definite, although alterations due to melanosis were found. Because of the racial pigmentation this was viewed as within normal limits but was carefully charted for later study. It is hoped that by follow-up the time of onset of definite changes due to alcaptonuria can be established. One author indicates that evidence of ochronosis may be found in the eyes as early as the third decade of life, although it is usually considered that the ochronosis is due to accumulation of pigment over a period of years and does not develop in alcaptonuria until middle age or later.

Owen C. Dickson.

Dimitry, T. J. **The socket after enucleation and the artificial eye.** Arch. of Ophth., 1944, v. 31, Jan. pp. 18-28.

The author defines enucleation as the peeling of the eye from Tenon's capsule. He gives a résumé of the various procedures for removal of the eyeball. He insists that random severance of muscles and tissues to rid the orbit of the globe is not enucleation but extirpation. In discussing what happens to the remainder of the contents of the orbit after enucleation, Dimitry states that it is readily demonstrable clinically that improvement does not follow the sewing of opposing rectus muscles but that, on the contrary, disfigurement of the socket is produced. Some men do not even sew the wound, and yet get excellent results.

Various modifications such as implants have been devised in an effort to obtain a mobile stump. Dimitry believes we accomplish the mobile stump but have been unsuccessful in obtaining the mobile prothesis. He is of the opinion that the plastic prothesis will largely solve the problem of a movable prothesis. He criticizes the ophthalmologist for turning the responsibility of the fitting over to a nonmedical technician. (8 figures, references.)

R. W. Danielson.

Ulluer, L. J. **Orbital cellulitis.** Rev. Oto-Neuro-Oft., 1942, v. 17, Aug., pp. 143-145.

The author considers orbital cellulitis an acute inflammation of the soft tissues of the orbit, characterized by bulbar congestion, lid edema and ptosis, limitation of ocular motility, chemosis, and a septic fever. He believes that the chief cause of orbital cellulitis is acute nasal-sinus infection (70 to 80 percent of all cases), the pathway being the sinus veins, thence into the superior and inferior ophthalmic veins. Swimmer's sinusitis and the acute exanthemata of childhood are frequent offenders.

Pathologically, orbital cellulitis may result in an orbital phlegmon or abscess, a subperiosteal abscess, or a thrombophlebitis with cavernous-sinus involvement. This latter must be differentiated early from orbital cellulitis. Treatment should be directed toward the infected sinuses (if they exist) drainage of the orbit, and chemotherapy.

Edward Saskin.

14

EYELIDS AND LACRIMAL APPARATUS

Jiménez González, R. **A new operative procedure for senile ectropion.** Arch. de la Soc. Oft. Hisp.-Amer., 1942, v. 1, Oct., pp. 339-344.

The method consists in the excision of a triangular piece of tarsus and muscle of the conjunctival aspect of

the lower lid. The base of the triangle is directed toward the fornix. A doubly-armed suture is inserted through the conjunctiva near the lateral incisions close to the base of the triangle, and the two ends of the doubly-armed suture, threaded on one needle, are then passed through the conjunctiva below the base of the triangle and brought out on the skin at about the center of the lower orbital margin. The threads are tied in this position, pulling them to the extent necessary to correct the ectropion. In subsequent dressings the position of the lid may be controlled by loosening or tightening the suture. (5 illustrations.) Ramón Castroviejo.

Lemere, H. B. **A vacuum device for the eyelids.** Arch. of Ophth., 1944, v. 31, Jan., p. 95.

Expression of the meibomian glands by digital pressure or by some form of spatula, while not very painful, is occasionally objected to by the patient. In a situation of this kind, the author attached the glass portion of a medicine dropper to the tubing of the suction apparatus used for nasal displacement treatment. He found that passage of the end of the dropper back and forth along the margin of the lids tended to evacuate accumulated material from both the meibomian glands and the hair follicles.

He has found this procedure a valuable adjuvant to treatment in practically all his cases of chronic and in some cases of acute conjunctivitis. He instills a drop of 0.5-percent solution of pontocaine hydrochloride a minute before using the suction, and two drops of a 1 to 500 solution of silver nitrate afterward to take care of any floating material resulting from the suction.

R. W. Danielson.

Rosen, Emanuel. **A case of acute metastatic dacryoadenitis.** Amer. Jour. Ophth., 1944, v. 27, March, pp. 276-278.

Thomas, Maxwell. **Management of chalazia.** Texas State Jour. Med., 1943, v. 39, Oct., p. 347.

Thomas shows a diagrammatic sketch of incisions for removal of chalazion. He discusses the etiology, diagnosis, and treatment and describes the procedure for thorough removal. Management of chalazia requires a knowledge of the anatomy of the lids, with a true conception of the pathologic process. Theodore M. Shapira.

15

TUMORS

Black, G. W. **Melanotic sarcoma of the iris.** Trans. Ophth. Soc. United Kingdom, 1942, v. 62, pp. 304-306.

Sarcoma of the iris furnishes 2 to 6 percent of the malignant tumors of the uveal tract. The author reports a case in a woman 64 years of age. A brown patch on the left iris which had been noticed for 40 years became slightly larger, with flattening of the pupillary margin. The mass was removed by iridectomy through a keratome incision and covered by a conjunctival flap. The pathologic report showed an early spindle-cell type of melanotic sarcoma, the peripheral portion intensely pigmented, corresponding to the brown patch. At the pupillary margin the tumor mass was highly cellular and contained scanty pigment.

Beulah Cushman.

Black, G. W. **Reticulum-cell sarcoma.** Trans. Ophth. Soc. United Kingdom, 1942, v. 62, pp. 316-317.

A plum-colored tumor attached to the outer surface of the external rectus

muscle by fibrous trabeculae was removed. On microscopic examination the bulk of the tumor consisted of large closely packed cells with rounded or oval vesicular nuclei and ill-defined cytoplasm as of reticulum cells. The arrangement was diffuse, with a fair number of mitotic figures. There was moderate infiltration by lymphocytes but no lymph follicles.

Beulah Cushman.

Burch, F. E., and Camp, W. E. **Results of irradiation of malignant melanomas of the uveal tract.** Trans. Amer. Acad. Ophth. and Otolaryng., 1943, 47th mtg., May-June, pp. 335-352.

Even intensive irradiation of the eye is valueless in any of the types of malignant melanoma. Roentgen therapy does not assure against metastasis; there is even a possibility that metastases develop earlier after irradiation than when it is not employed. Irradiation should be reserved for cases in which only the tumor-containing eye retains vision, for cases in which the patient refuses enucleation, and those in which the orbit and adjacent structures are already invaded. (4 tables.)

Gertrude S. Hausmann.

Hollander, L., and Krugh, F. J. **Cancer of the eyelid.** Amer. Jour. Ophth., 1944, v. 27, March, pp. 244-253. (22 figures.)

16

INJURIES

Allen, T. D. **Industrial ophthalmology.** Surg. Clinics North America, 1942, v. 22, Aug., pp. 1011-1028. (See Section 18, Hygiene, sociology, education, and history.)

Berens, C., and McAlpine, P. T. **Solar keratoconjunctivitis associated with amblyopia.** Amer. Jour. Ophth., 1944, v. 27, March, pp. 227-231. (6 fields, references.)

Dickson, R. M. **First-aid treatment of industrial eye injuries.** Brit. Jour. Ophth., 1943, v. 27, Dec., pp. 544-548.

The author records the results after six months trial of the use of 10-percent sodium-sulphacetamide solution as first-aid treatment in eye injuries in 30 mines and 30 factories. Of the total injuries in 30 mines in Scotland, 96 percent returned to work with no loss of working time. In the shipyards 98.87 percent of the patients lost no working time. (References.)

Beulah Cushman.

Gundersen, T., and Liebman, S. D. **Effect of local anesthetics on regeneration of corneal epithelium.** Arch. of Ophth., 1944, v. 31, Jan., pp. 29-33.

Clinically it has been observed from time to time that local anesthetics have an inhibitory influence on the regeneration of corneal epithelium. In this investigation, the corneas of guinea pigs were abraded and measurements were made of length of time required for regeneration of the corneal epithelium when various anesthetics were used hourly during healing. The drugs used were solutions of 10-percent cocaine hydrochloride, 4-percent cocaine hydrochloride, 1-percent butacaine sulfate, 4-percent larocaine hydrochloride, 1-percent phenacaine hydrochloride, and 0-5-percent tetracaine hydrochloride. All solutions contained 0.5-percent chlorobutanol.

Both 10- and 4-percent cocaine very definitely retarded the regeneration of epithelium; more retardation was observed with the stronger solution. In fact, repeated instillations on the intact epithelium produced corneal areas

which stained with fluorescein. The inhibitory effect of cocaine diminished as increasingly hypertonic solutions were used. Tetracaine gave similar results, although less pronounced. The delayed healing of the epithelium was observed in varying degrees with 1-percent phenacaine, 1-percent butacaine sulfate, and 4-percent larocaine. One percent phenacaine and 0.5-percent tetracaine were the least toxic. The pH is probably not a determining factor, since buffered solutions at a pH comparable to that of the anesthetics used showed relatively little inhibitory action. Two very descriptive charts are included. (References.)

R. W. Danielson.

Hughes, W. F. **Management of injuries to the eyes.** Surg. Clinics North America, 1942, v. 22, Oct., pp. 1355-1373.

The author writes especially in regard to war injuries. The article is essentially a résumé of the diagnosis and treatment of various injuries, including lacerations, fractures of the bones of the orbit, intracorneal foreign bodies, perforating injuries, concussion injuries to the eye, and burns, especially those due to war gases. (References.)

R. C. Richardson.

Jaime, G. G. **Cephalic tetanus of ocular origin.** Rev. Oto-Neuro-Oft., 1942, v. 17, Jan.-Feb., pp. 22-26; March-April, pp. 52-58; May-June, pp. 88-94.

Cephalic tetanus, or tetanus limited to the region of the head, follows facial injury (orbit, nose, temporal, or malar regions). There is an incubation period of at least one week, after which the typical unilateral or bilateral trismus occurs, followed later by facial paralysis. This paralysis is of the peripheral type, and the muscles involved need not be near the site of injury. Occasionally, the above symptoms are preceded by an ophthalmoplegia manifested by ptosis. Other eye signs noted are miosis and fixed pupils. The author cites several cases of cephalic tetanus following ocular injury, such as perforation of the cornea associated with loss of the eye. The affection is relatively rare. Etiologically, the offending organism is Nicolaier's bacillus, clostridium tetani, found in the infected wound in or around the lids, orbit, or the eyeball. The author claims conclusive proof that the organism is Nicolaier's bacillus, and that there is an associated virulent toxin. He offers a mechanical theory of the pathogenesis by which the toxin causes a direct nerve irritation. A reflex theory of the pathogenesis involves the trigeminus as the conducting pathway of the toxin, which latter affects the facial nerve and the third, fourth, or sixth nerves. A third, directly infectious theory claims a toxic or bacterial invasion of the body fluids from the infected wound. The author states that whatever theory is finally accepted the passage of the organism or toxin is centripetal, backward from the wound.

The author discusses his studies of diagnosis, course, prognosis, symptomatic and specific treatment, active and passive immunization, preventive serotherapy, therapeutic serotherapy, and anaphylaxis. The diagnosis is simple, given an infected wound about or of the eye, an incubation period, then facial trismus or paralysis. The course may be acute, subacute, or chronic, lasting as little as two days and ending in death, or becoming protracted with a more satisfactory outcome. The prognosis is usually guarded, even in the protracted cases. There may be residual paralysis or paresis if death does

not intervene. The treatment is symptomatic, as in generalized tetanus. Specific antitoxin is administered in addition. The author's active immunization is similar to our vaccination with an attenuated toxin; passive immunization is specific. He also discusses using blood serum from well individuals previously stricken, as well as intravenous, intra-arterial, and intracranial administration of the antitoxin, always being on the watch for hypersensitivity. Edward Saskin.

Kirby, D. B., and Town, A. E. **Injuries of the eyes and eyelids.** Surg. Clinics North America, 1943, v. 23, April, pp. 404-438.

The authors present a comprehensive discussion and review of the prevention, diagnosis, and treatment of ocular injuries of all kinds. The usual treatment of traumatic lesions and burns is covered, as well as later surgical treatment—plastic surgery—to remedy defects. (10 illustrations, references.) R. C. Richardson.

Mann, Ida. **A study of epithelial regeneration in the living eye.** Brit. Jour. Ophth., 1944, v. 28, Jan., pp. 26-39.

Experimental study of epithelial regeneration in the eyes of rabbits showed formation of pigment in the basal cells of the conjunctival or corneal epithelium. The pigment allowed recognition of the particular cells involved, and their migration could be followed from day to day with the loupe or slitlamp. The initial loss of epithelium was confirmed by the use of fluoresceine, and the observations originally made on rabbits were confirmed clinically in man. The injuries were produced by scraping, chemicals and heat, without damage to the underlying tissues.

The first series of experiments demonstrated the movement of the pigmented epithelium in an irregular line which met the slide of epithelium from the opposite side in less than 20 hours. The pigment from these advanced cells gradually disappeared as the cells were replaced, and the original pigment line, which had become thinner, gradually assumed its original density. The area of spread from the activating substance seemed to be more than 2.5 mm. and less than 5 mm. from the edges of the wound in all directions. If the injury was produced by a hot wire the movement of the pigment epithelium was much less, was very irregular, and did not occur all around the burned area, the heat having evidently destroyed the activating substance. If the conjunctiva was injured near the cornea, the pigmented cells slid toward the area as though the wound were in the cornea.

A second series of experiments involved injury to larger areas of the cornea by various chemicals. Healing of the superficial loss was usually complete within four days, whereas the pathologic process continued its course beneath the epithelium for days or weeks. If the whole width of the ring had migrated, the gap in the limbal pigment never completely regenerated.

A third series of experiments dealt with production of pigment proliferation in response to chemical injury by the arsenical war gases. In this type of injury the proliferation did not occur until after healing of the epithelium, but it was progressive and continued to increase for months.

A fourth series of experiments dealt with pigment migration in vitamin-A deficiency. It could be produced experimentally in rabbits without epithelial loss, and was based upon a wandering of chromatophores.

The experiments corroborate the general agreement that epithelium heals by migration from the edge of the denuded area, associated with mitosis at some distance from the defect. Thus the loss is made good by pre-existent cells which move actively across to cover the raw zone, and these cells are replaced by cell division behind their original positions and not over the wound area. (14 illustrations, references.) Beulah Cushman.

Moore, P. G. **Management of magnetic foreign bodies in the eye.** Ohio State Med. Jour., 1944, v. 40, Jan., p. 26.

An adequate headrest, oblique illumination, a loupe with a half-inch focal distance, and a sharp Graefe knife are stressed by the author as necessary for proper removal of corneal foreign bodies. Distinction between the dead white ring of coagulated tissue surrounding a hot foreign body and the gray of an infected infiltrate is important. The author uses a 1 to 500 zinc sulphate solution as routine antiseptic. He feels that bandaging of eyes following removal of obviously uninfected foreign bodies is not indicated if the corneal surface is not denuded. Infection, shown by the appearance of a gray ring on the second day, requires wiping out the wound with a stronger antiseptic such as 3.5-percent iodine or trichloracetic acid.

Management, indications for anterior or posterior approach, and postoperative care of patients with intraocular magnetic foreign bodies are also discussed. Indications for enucleation are enumerated, with the emphasis on conservatism unless the eye is unquestionably blind and irritable or painful.

Owen C. Dickson.

Pierse, Dermot. **A case of removal of nonmagnetic foreign body from the ciliary region.** Brit. Jour. Ophth., 1943, v. 27, Dec., pp. 550-552.

In a man 38 years of age a small piece of brass perforated the cornea and was visible in the angle of the anterior chamber. The vision was 6/9, the lens clear, and no abnormality was seen in the fundus. There was no response to the giant magnet, and the fragment disappeared backward upon the attempt to grasp it through a corneal incision. It was then localized accurately on an X-ray film with the aid of a needle inserted in the sclera. The approach was made 4.0 mm. from the limbus, using a hinged flap of sclera. The ciliary body was investigated with the point of the knife until the foreign body was felt, when it was easily removed through the ciliary body. The scleral flap was closed with a previously inserted suture, and the conjunctiva replaced. Two weeks later the vision with −0.75 cyl. axis 175° was 6/6. (References.)

Beulah Cushman.

Robson, J. M. **Experimental corneal ulcers.** Brit. Jour. Ophth., 1944, v. 28, Jan., pp. 15-25. (See Section 6, Cornea and sclera.)

Rosen, Emanuel. **An interesting case of rupture of the choroid.** Brit. Jour. Ophth., 1943, v. 27, Dec., pp. 552-554.

The author reports two cases with rupture of the choroid in the macular area, one with resulting corrected vision of 20/20 and the other of 8/200. He concludes that the distinct anatomy of the macula modifies the traumatic pathology. (2 illustrations.)

Beulah Cushman.

17

SYSTEMIC DISEASES AND PARASITES

Carmichael, F. A. **Migraine, a review.** Jour. Kansas Med. Soc., 1943, v. 44, Oct., p. 333.

The basic mechanisms mentioned are: pressure on cranial and upper cervical nerves, traction on or spasm of the basilar vessels, traction or reduced caliber of venous sinuses, and inflammation in or about pain-sensitive structures. Causation involves many apparently unconnected details which, however, can be resolved into three fundamental elements combined in varying proportions. The first is the hereditary factor, which is known to exist in 70 percent of migraine sufferers and which includes a predisposition to this syndrome in some parts of the brain and of the autonomic nervous mechanism. The chemical details of these changes are not yet known, but those afflicted frequently also have other neuroses especially involving the vasomotor mechanism. The second factor is that of age, 80 percent of migraine beginning between the ages of eight and ten years, somewhat before puberty. Most women sufferers are relieved by the menopause, and in some the attacks are influenced by menstruation. The third or environmental factor includes physical and chemical conditions of many kinds which intensify or retard the hereditary predisposition, thus altering the minimal time between attacks as well as their severity. Among those mentioned are histamine sensitiveness, the environmental factors which aggravate hypertension, compression of the fifth nerve, and a small calcarine tumor whose removal was curative. Migraine, however, is apparently less frequent in patients with liver or biliary tract disease, the reason being as yet unknown.

In the treatment two drugs are emphasized: ergotamine tartrate, a vasoconstrictor, and potassium thiocyanate, a vasodilator. The former should not be used in patients with anginal symptoms and other evidence of obliterative vascular disease. The latter is apparently better adapted for migraine sufferers with hypertension.

Charles A. Bahn.

Garcia Miranda, A. **Avitaminosis in ophthalmology.** Arch. de la Soc. Oft. Hisp.-Amer., 1942, v. 1, Oct., pp. 344-377. (See Section 2, Therapeutics and operations.)

Greenberg, M. M. **The ocular manifestations of multiple sclerosis.** Dis. Eye, Ear, Nose, and Throat, 1942, v. 2, Oct., p. 296.

A comprehensive summary is given of the history, symptoms, course, etiology, and treatment of multiple sclerosis, with detailed attention to the ocular manifestations.

Robert N. Shaffer.

Harris, Wilfred. **Ataxic nystagmus. A pathognomonic sign in disseminated sclerosis.** Brit. Jour. Ophth., 1944, v. 28, Jan., pp. 40-42. (See Section 4, Ocular movements.)

Lloyd, R. I. **Less evident causes of lowered acuity in senility.** Amer. Jour. Ophth., 1944, v. 27, March, pp. 232-243: also Trans. Amer. Ophth. Soc., 1943, v. 41. (13 illustrations, references.)

Michaelson, I. C. **Ocular manifestations of neuroses found among soldiers.** Brit. Med. Jour., 1943, Oct. 30, p. 538.

This report deals with the ocular manifestations of hysteria and of the

chronic anxiety state, these being the two most common neuroses. In hysteria, the most common symptoms are diplopia and defective day or night vision.

Of the anxiety states, asthenopia, headache, photophobia, muscae volitantes, and epiphora are more common. The authors point out the importance of evaluating functional and organic components in each case. In any of the more severe cases the help of a psychiatrist is needed to effect the profound personality change necessary for permanent removal of the underlying cause of the patient's symptoms.

Robert N. Shaffer.

Murphy, R. C., Jr. **An eruptive fever involving the mouth and eyes (Stevens-Johnson disease).** New England Jour. Med., 1944, v. 230, Jan. 20, p. 69.

About twenty cases characterized by acute and at times fulminating systemic reactions with fever, prostration, generalized skin rash, and severe stomatitis followed by sloughing with eventual recovery have been reported. A mucopurulent type of conjunctivitis is noted almost constantly. No etiologic agent has been discovered. Panophthalmitis has occurred with loss of the eyes in several patients. This may be due to secondary conjunctival invasion by staphylococcus aureus as suggested by recovery of this organism in the present reported case. Previously reported cases ran from 22 months to 16 years, the great majority being in males.

The present case is that of a 22-year-old Italian male. At onset the disease appeared as a tenderness of the buccal mucosa, with rapid appearance of tight whitish vesicles surrounded by erythema, which proceeded to rupture and slough. Temperature between the third and sixth days varied around 104.6 F. No vesicles appeared on the conjunctivas, although they were diffusely inflamed and a mucopurulent discharge was present. The temperature fell on the eighth day. Similar lesions (vesicles) appeared on the extremities and on the glans penis and corona. Crusting of the lesions had occurred by the seventeenth day, and before the end of the fourth week the scars were barely visible.

This is thought to be a definite clinical entity, although etiologic relation to Vincent's stomatitis is not confirmed. The description used for these cases has been "erythema multiforme bullosum with involvement of the mucous membrane of the eyes and mouth."

Owen C. Dickson.

Negus, V. E. **The relationship of ophthalmology and rhinology.** Brit. Jour. Ophth., 1943, v. 27, Dec., pp. 554-557.

The author urges that in many conditions about the nose and eyes the rhinologist and ophthalmologist should work together. He suggests that the label "orbital cellulitis" is often misleading, these being really cases of frontal sinusitis and acute ethmoiditis with orbital edema. They should be treated with lavage of the maxillary sinus, which is usually infected, and with an ephedrine spray.

The author has seldom found iridocyclitis due to suppuration of the nasal sinuses, although infected tonsils were often causative factors. Desensitization and medical treatment of nasal allergy often improved a concurrent conjunctivitis.

Beulah Cushman.

Thygeson, Phillips. **Virus diseases of the eye.** Trans. Pacific Coast Oto-Ophth. Soc., 1942, v. 27, pp. 58-102.

The material covered by this article is very similar, although somewhat more briefly stated, to that published in the Archives of Ophthalmology, 1943, v. 29, Feb., p. 285; March, p. 488; and April, p. 635. (See abstracts, Amer. Jour. Ophth., 1943, v. 26, June, p. 666; and Aug., p. 892.)

Woods, A. C. **Syphilis of the eye.** Amer. Jour. Syph., Gonorrhea, and Ven. Dis., 1943, v. 27, March, p. 133.

This long and valuable survey of the subject includes consideration of the various stages of congenital and acquired syphilis respectively, with a statement of the lesions which prevail in each period of either form of the disease. Consideration is given to the type of treatment suitable for each combination. Particularly valuable sections are those dealing with neuro-ocular lesions of late meningovascular neurosyphilis, and with degenerative neurologic lesions. Three basic types of optic-nerve atrophy are discussed, with the corresponding visual fields. Type one, representing 12 percent of these cases, progresses from the periphery into the center, and has concentric visual fields and late loss of vision. Type two (34 percent of the cases) progresses from the pia in the form of a wedge, involves sectors of the optic nerve, and has sector-shaped fields and either early or late loss of vision. Type three (54 percent of the cases) progresses as a generalized degeneration, and has cecocentral scotomas, either with or without peripheral field defects, and with early loss of vision. We are reminded that vitamin-A and B deficiencies may be contributory or aggravating factors. Subdural treatment with arsphenaminized serum is said to reduce blindness to 50 percent at the end of nine years but is occasionally responsible for sudden extinction of what vision is present. For malaria and other forms of fever therapy it is claimed that only 14 percent of the patients are blind at the end of nine years. Charles A. Bahn.

Yudkin, A. M. **Nutrition as it affects the eye.** Med. Clinics of North Amer., 1943, v. 27, March, pp. 553-560.

Brief consideration is given to ocular disturbances on a background of nutritional deficiency, with special mention of night blindness, conjunctivitis and blepharitis, chorioretinitis, and vitamin therapy. The author also discusses secondary ophthalmic disorders which are regarded as sometimes responding to treatment with vitamins, such disorders including marginal ulcers of the cornea, early stages of cataract, hemorrhagic retinitis, and toxic amblyopia from alcohol and tobacco. (References.)

W. H. Crisp.

18

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Allen, T. D. **Industrial ophthalmology.** Surg. Clinics North America, 1942, v. 22, Aug., pp. 1011-1028.

Allen discusses industrial ophthalmology from the standpoint of the general surgeon, rather than from the standpoint of the ophthalmologist. The importance of preëmployment and periodic eye examination is considered, as well as that of good lighting in industry. There is mention of the treatment of minor eye injuries, as well as of the treatment of more serious injuries such as retinal detachment, intraocular foreign body, and ocular burns. (7 illustrations.) R. C. Richardson.

Beach, S. J. **The American Board of Ophthalmology learns about written examinations.** Med. Clinics of North America, 1943, pp. 1409-1415. (See Amer. Jour. Ophth., 1943, v. 26, Sept., p. 911.)

Birren, F. **Color technique in industry.** Sight-Saving Review, 1943, v. 13, May, p. 3.

Selection of colors in industry must be directed at minimizing work hazards and ocular fatigue. Rest rooms, lunch rooms, and so on, should feature soft, cheerful colors—as blue and rose. In work rooms, ceilings and upper walls should be white or bright blue; floors, a light color reflecting about 25 percent; and lower walls and machinery, a soft gray color reflecting 25 to 40 percent—thus giving the fixated part of the field the same illumination as the adjacent parts.

In general, yellow (not red) should be used for critical hazards; yellow-green or orange for lesser hazards; and red reserved for fire escapes, exits, and the like. Benjamin Milder.

An eye health program for schools. Sight-Saving Review, 1943, v. 13, May, p. 22.

The minimum recommended intensity of illumination for close work at school should be 15 foot-candles, in diffuse, glare-free illumination. Natural illumination should be provided through windows occupying space at least equivalent to 16 percent of the area of the floor, situated to the left of the pupils. Walls should be of light color, with dull finish. Seating should be so arranged that the desks are turned at a 30° angle away from the windows. (The authors do not seem to give any attention to the question of the teacher's position in relation to the light, or of reflections from the blackboard.) Printed matter should be 10-point for children over twelve years old, increasing to 24-point or 30-point for children under seven years.

Education of teachers is important in an eye health program, with emphasis on special attention to those children requiring it. The testing of visual function must be undertaken in school. Cooperation between parents, school, and medical profession will produce the best results. Education of child, parents, and the community is the final, all-important objective of an eye health program for schools.

Benjamin Milder.

Gruber, K. F. **The vocational training program for the visually handicapped in the Minneapolis public schools.** Sight-Saving Review, 1943, v. 13, no. 2, p. 104.

Before the establishment of a vocational-training program the visually handicapped were graduated from the Minneapolis public schools with purely academic skills. The present plan permits 12th-grade students in the sight-saving classes to spend part of their time in selected industries earning credits to be applied toward graduation.

Careful studies of aptitude of individual pupils are made through cooperation of an ophthalmologist, a psychologist, and the school, including a coördinator between school and industry. The family background is studied; intelligence and aptitude tests are given; recommendations are made. The student chooses the training plan which interests him most.

In the industry the pupils are carefully supervised. Every effort is made to make them compete on even terms

with nonhandicapped workers. In the school, effort is made to coach the pupils in qualities, such as punctuality, courtesy, independence, and personal appearance, which will aid them in obtaining employment.

Robert N. Shaffer.

Kuhn, H. S. **Significance of visual defects in war production effort.** Jour. Amer. Med. Assoc., 1943, v. 123, Dec. 25, p. 1085.

The consulting ophthalmologist working with industries must supervise with extreme vigilance the entire industrial program; treatment of eye injuries, eye-protection plans, job standard evaluations, and rehabilitation procedures. Physicians should not hesitate to offer their services to industries. (One figure, 2 tables, discussion.)

George H. Stine.

Landa Lion, Luiz. **Ophthalmology of high altitude.** Arquivos Brasileiros de Oft., 1943, v. 6, Aug., pp. 141-143.

A special ophthalmology of high altitude exists by reason of the special meteorological, climatic, and racial conditions. The author finds in the South American altiplane a people with special characteristics, religious, artistic, and physical. These people have large thorax and slight development of the lower extremities. Their physiologic attributes include hypotension and polyglobulia. A semianesthetic condition of the tissues arises from the chewing of coca by 70 percent of the population. Glaucoma is rare, and the author speaks of this fact as supporting the interpretation of that disease as a local manifestation of a general condition. Pterygium is frequent in Bolivia as compared with other countries, and is interpreted by the writer as a mechanism of defence against the constant action of the winds occurring in that region. In La Paz trachoma is unknown, and the trachoma patients of Argentina visit that city for relief from their disease. It is said that, on account of the chewing of coca, the Bolivian ophthalmologist, when operating upon the eye, does not have to concern himself greatly with the question of anesthesia.

W. H. Crisp.

Lester, Eileen. **Opportunities for the visually handicapped through job analysis.** Sight-saving Review, 1943, v. 13, May, p. 29.

The visually handicapped may fall in this category by virtue of low corrected visual acuity, impairment of visual field, progressive ocular disease, various degrees of color blindness, muscular imbalance, or impairment of stereopsis.

Industrial surveys indicate that the vast majority of large concerns make no effort at job analysis in regard to visual requirements; a majority make an inadequate visual test, or none at all; and very few have any program for conservation of sight.

It is essential to undertake thorough job analysis to determine what jobs can be performed by persons with visual limitations. It is equally important to provide a full and adequate program of examination of all job applicants, including the visually handicapped, with an estimate of how the individual can best use whatever visual abilities he has.

Benjamin Milder.

Mann, Ida. **The scope of prevention in ophthalmology.** Brit. Med. Jour., 1943, Oct. 16, p. 482.

The existence in England and Wales of 74,000 blind persons whose support costs the state 4½ million pounds yearly emphasizes the importance of planned prevention of blindness. Re-

duction of the incidence of eye diseases could come from three ways: (1) Improving the services of the oculist, by making modern ophthalmic-hospital treatment available to everyone, by standardizing the system of training ophthalmologists and requiring a uniform standard of examination, by close supervision and better facilities for diagnosis and treatment of children under school age and of adolescents leaving school. (2) Educating the public. Some biologic and physiologic instruction in schools should be obligatory, including the mechanism of sight, the errors of refraction, and presbyopia. The industrial worker should be instructed in methods of protecting the eyes. (3) Establishment of research teams (in association with laboratories) in which physicists, biochemists, physiologists, and pathologists among others could unite with ophthalmologists for solving given problems.

R. Grunfeld.

Martin, H. G. **Fads and fallacies regarding our eyes.** Milwaukee Med. Times, 1943, v. 16, Nov., pp. 15-19.

As 80 percent of our intellectual development is acquired through visual processes, and as our eyes were not intended by nature to be used as we use them, the symptoms of ocular discomfort are frequent. The author does not, however, discuss in detail just how to avoid the misuse of the eyes. Ocular conditions stand first or second on all lists as causes of rejection for the armed forces. Clear sight develops at the end of the first year and binocular vision about the fourth year. The author believes that faulty ocular habits during this time produce symptoms later in life, and that many children's eyes are not given a chance to develop normally. Such exercises to strengthen the eyes as are now in vogue have had periods of favor during the past hundred years, Rolling the eyes in different directions, as popularly practiced, exercises strong and weak muscles equally, and therefore can accomplish no good.

Charles A. Bahn.

Morgan, D. H. **Vocational aptitudes of the visually handicapped.** Outlook for the Blind, 1943, May, p. 125.

Advanced vocational training for the blind and partially blind is discussed. Examples of successful candidates from the California School for the Blind for placement in the shops of the public high schools are described. A program for handling the visually handicapped is offered, with special suggestions on how to overcome the discouraging attitude of principals of public high schools.

Francis M. Crage.

Prado, Durval. **Contribution to the history of optics in São Paulo.** Arquivos Brasileiros de Oft., 1943, v. 6, Aug., pp. 124-127.

The history of optics in São Paulo, Brazil, dates back about a half century. The first of three phases was purely commercial. Most of the glasses were of French origin and took the form of pince-nez. At that time, only sufferers from external disorders of the eyes made free use of smoked or blue glasses. The choice of glasses depended entirely at best upon a short reading at the time of purchase, the person's age serving as the main criterion for determining the proper "number." Systematic borrowing of glasses among the less fortunate was frequent. The glasses were graded by numbers in inches. They were sold at stores for surgical supplies, musical articles, clocks, and so on.

The first attempt at an optical work-

shop appeared about 1895, the grinding being done on a stone wheel moved by a pedal managed by the operator. Up to that time São Paulo had not possessed physician oculists properly so called. The few prescriptions which appeared had to do with presbyopia or simple myopia.

Beginning with 1902 the scientific phase of optics in São Paulo appeared with the settlement there of a number of physician oculists, foreign and Brazilian, almost all of whom had had special courses in Europe. Up to 1912 prescriptions for myopia predominated, although Pignatari began in 1902 the prescription of cylindrical lenses and the notation in diopters. The cylinders were usually prescribed at horizontal or vertical axes. But some prescriptions showed axes between 30 and 60 degrees. Beginning with 1912, the newly founded School of Medicine and Surgery in São Paulo taught ophthalmology as a true specialty. The laws of Brazil have not yet recognized a profession of optometry. W. H. Crisp.

Suheyl Unver. **Remarks concerning the history of Turkish ophthalmology.** Göz Klinigi, 1943, v. 1, Aug., p. 16.

In the Orient ophthalmology was practiced as a specialty for many centuries, especially in Turkey. The ophthalmologist was called "Kehhal," which is Arabic and originally meant "cosmetic for the eye." Reference is made to many old textbooks and monographs about eye diseases, generally written in the Arabic language, but some of them in Turkish. The most famous authors were Ali Ibni Isa Elmedujisi (ninth century) and Ibni Sina (Avicenna, 980-1037 A.D.). Centuries ago an eye doctor was attached to each hospital. J. Igersheimer.

Vaughan, M. S. **Public health nursing in a trachoma control program.** Sight-Saving Review, 1943, v. 13, no. 2, p. 91.

The author summarizes the methods and difficulties of establishing a trachoma-control clinic in Arkansas, organizational structure and efforts to educate both personnel and public being outlined. She sums up her article by saying that "the public-health nurse's services in a trachoma-control program are essentially the same as in any other public-health program for control of communicable disease."

Robert N. Shaffer.

Weiss, Charles. **The geographical distribution of ocular infections.** Amer. Jour. Ophth., 1944, v. 27, Feb., pp. 175-177. (One table, references.)

19

ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

Stone, L. S. **Return of vision in transplanted adult salamander eyes after several days of refrigeration.** Proc. Soc. Exper. Biol. and Med., 1943, v. 34, Oct., p. 44. (See Section 13, Eyeball and orbit.)

Stone, L. S., and Dinnean, F. L. **Lens induction in the salamander (amblyostoma punctatum) with special reference to conditions in experimentally produced cyclopia.** Yale Jour. Biol. and Med., 1943, v. 16, Oct., p. 31.

In a series of experiments the authors show that the lens in amblyostoma punctatum arises in the surface ectoderm by induction of the eye-forming center at a definitely circumscribed period in its development. If this center is absent, no lens is produced. Defects

produced in the prechordal substrate by immersion in lithium-chloride solution or by surgical means gave rise to cyclopia and other malformation. This emphasizes that conditions in the lens are always subservient to the dominance of the eye-forming centers.

Robert N. Shaffer.

PAN-AMERICAN NOTES

Edited by Dr. M. Uribe Troncoso
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Communications should reach the Editor by the twelfth of the month

Miscellaneous

Argentina. Under the direction of Prof. Baudilio Courtis the Section of Ophthalmology of the "Patronato Nacional de Ciegos" has been entirely reorganized. This institution for the blind opened, one year ago, a modern dispensary called "Consultorio Central—'Pedro Lagleyze'" in honor of Professor Lagleyze, one of the most famous oculists of Buenos Aires. The building of the Patronato was erected by private contributions amounting to $150,000. It is a large structure of modern architecture, has four floors, and is provided with complete appliances for the diagnosis and treatment of eye diseases.

Brazil. In July of 1943, a decree was signed instituting the compulsory use of Créde's method in the State of Bahia.

Journals

Anales de la Sociedad Mexicana de Oftalmologia y Oto-Rino-Laringologia. This journal contains the following articles dealing with ophthalmologic subjects: July and August, 1942, Dr. José Saenz Canales: "Cystic formations in the vitreous, a clinical case"; September-October, 1942, Dr. M. de Rivas Cherif: "Bio-photo-micrography of the eye—A preliminary note"; November and December, 1942, Dr. Paul A. Chavira: "Treatment of penetrating injuries of the anterior segment of the eye"; May-August, 1943, Dr. Paul Gonzáles Enríquez: "Optochiasmatic meningitis," with a commentary by Dr. Manuel Marquez; and September-December, 1943, Dr. Robert Fitzpatrick: "Mydriasis and the different drugs to produce it."

Societies

Argentina. At the third monthly meeting, held in May, 1943, The Argentinian Ophthalmological Society awarded its annual prize for "the best paper read" to Drs. Flaminio Vidal and Jorge Malbran for their essay on "Disposition of myelin fibers in the chiasma of a cat." (Experimental study.)

The Ophthalmological Society of the Litoral in Rosario, Argentina, elected the following officers to serve during 1942-1944: President, Prof. Carlos Weskamp; General Secretary, Prof. Adj. Dr. Juan Manuel Vila Ortiz; Treasurer, Prof. Adj. Dr. Roberto E. Giqueaus; Members: Prof. Adj. Dr. Isaac Cottlier, Dr. Luis A. Gallo, Dr. Salvador Imbern, and Dr. Juan Maggi Zavalia.

II Pan-American Congress of Ophthalmology. The Uruguayan Committee in charge of the organization of the Congress, taking into account the world situation, feels that the meeting should be postponed until November 3, 1944. The scientific program remains unchanged, one day being given to each of the following four sections; as follows:

Social Ophthalmology: Official papers prepared by the Committees on Prevention of Blindness and Trachoma of the Pan-American Congress of Ophthalmology.

Research Papers—Medical Ophthalmology: Four papers on glaucoma have been chosen for this section as follows: "The pre-glaucomatous state—Its diagnosis and treatment." "New ideas on glaucoma derived from gonioscopy." "Estimation and mechanism of the destructive effects of ocular hypertension." "Surgical intervention in glaucoma—How far can medical treatment be continued?"

Surgical Ophthalmology: "Surgical treatment of paralytic strabismus." "Surgical treatment of heterophorias." "Surgical treatment of heterotropias."

Free Papers.

Chile: The Chilean Committee of the Pan-American Congress of Ophthalmology has elected the following officers: President, Prof. Carlos Charlin; Vice-Presidents, Prof. C. Espildora Luque, Prof. I. Martini, and Dr. S Barrenechea who is the Chilean Delegate to the Council of the Congress; Treasurer, Dr. Ida Thierry; Secretary, Prof. J. Vardaguer; Members of the Committee, Drs. A Schweitzer, R. Costa, and R. Contardo. Dr. J. Thierry is the president of the Valparaiso Committee and the members of this committee are Drs. Tuyl Kuhlman, and A. Robert. The Chilean Com-

mittee has already sent a list of subscribers to the Pan-American Congress of Ophthalmology to the executive secretary, Dr. Conrad Berens.

A new society of the Fellows of the Kellogg Foundation—Pan-American Congress of Ophthalmology. On October 13, 1943, at a meeting in Chicago at which were present Drs. Bertha R. Noble, Daniel Silva, and Eduardo Barduño, from Mexico; Manoel da Silva from Brazil; Carlos H. Candray from El Salvador; Juan A. Diaz, from the Dominican Republic; Gustavo Vasquez from Paraguay and Augustin Peret from Venezuela, Fellows of the Kellogg Foundation Pan-American Congress of Ophthalmology, it was decided to found a Society which will promote a greater understanding between the Kellogg Fellows and develop cultural and social exchange between the ophthalmologists of the Americas. Dr. Harry S. Gradle was nominated honorary president in recognition of his work in Pan-American ophthalmology. A committee composed of Drs. Daniel Silva, Augustin Peret, and Manoel da Silva was elected to organize the Society and to draw up the statutes.

Personals

At an extraordinary public meeting on July 29th, Dr. Romulo Gil was received as a member of the Argentine National Academy of Medicine. Dr. Gil will take the place of Dr. Noceti, deceased.

The Moura Brasil Prize for 1943 was won by Dr. Sylvio de Abreu Fialho of Rio de Janeiro, with a paper on "Contribution to the problem of the diagnosis of early glaucoma."

The following is a list of the doctors who are already in the United States studying at selected clinics as beneficiaries of the Kellogg Foundation–Pan-American Congress of OphthalmologyFellowships: *Brazil:* Dr. Manoel Antonio da Silva of São Paulo, at the Illinois Eye and Ear Infirmary, Chicago; Dr. Heitor da Costa Pinto Marbach of Bahia, at The Johns Hopkins Hospital, Baltimore; Dr. Geraldo Queiroga of Minas Geraes, at the University of Iowa, Iowa City. *Chile:* Dr. Michel Mehech Haddad of Santiago at the Mayo Clinic, Rochester, Minnesota. *Costa Rica:* Dr. Alvaro Montero Padilla of San José at Cook County Hospital, Chicago. *The Dominican Republic:* Dr. Juan Antonio Diaz Espinal, of Ciudad Trujillo, at Wills Hospital, Philadelphia. *El Salvadore:* Dr. Carlos Candray of San Vicente, at the Massachusetts Eye and Ear Infirmary, Boston. *Haiti:* Dr. Joseph Roger Malebranche of Cap-Haiten, at New York University, New York City. *Honduras:* Dr. Efrain Ochos Reina of Comayaguela, at the Memphis Eye, Ear, Nose and Throat Hospital, Memphis. *Mexico:* Dr. Eduardo Garduño Ballesteros of Villa Obregón, at the New York Eye and Ear Infirmary, New York City; Dr. Daniel Silva Lopez Hermosa of Mexico City, at Northwestern University, Chicago; Dr. Bertha Riveroll Noble of Mexico City, at Tulane University, New Orleans. *Paraguay:* Dr. Gustavo Adolfo Vazquez of Asunción, at Columbia University, New York City. *Peru:* Dr. Enrique Samuel Haro of Lima, at the Stanford Lane Hospital, San Francisco. *Puerto Rico:* Dr. Mariano Cecil Caballero of San Juan, at the New York Eye and Ear Infirmary, New York City. *Venezuela:* Dr. Agustín Perret-Gentil of Caracas, at the Illinois Eye and Ear Infirmary, Chicago.

Two have already completed their Fellowships and returned to their homes. These are: Dr. Alexander W. Arathoon of Guatemala, who had a Fellowship at the Massachusetts Eye and Ear Infirmary, and Dr. Francisco de Almeida Rosa of São Paulo, Brazil, who studied at the University of Michigan and the New York Eye and Ear Infirmary.

Drs. Andreine Campanella and Maria C. de Carvalho have been appointed by the Institute of Inter-American Affairs to take an ophthalmologic course at the Washington University, St. Louis, Mo.

During the last two weeks of November, Prof. Moacyr E. Alvaro, executive secretary of the Pan-American Congress of Ophthalmology, visited the Argentine and Uruguay. In Buenos Aires, Dr. Alvaro was invited by Dr. Malbran, president of the Argentine Society of Ophthalmology, to preside at the annual meeting of that Society, where Dr. Sená read a paper on "Sulfonamides in ophthalmology." At the Alvear Hospital, at the invitation of Dr. Belgeri, Dr. Alvaro lectured on "How can the ophthalmologist cooperate with the general practitioner in order to obtain a correct diagnosis?" and at the Rawson Hospital at the invitation of Dr. Carlos Damel, head of the Ophthalmological Department of that hospital, Dr. Alvaro gave another lecture on "The advantages of organized ophthalmology." Dr. Alvaro took the opportunity offered by this trip to visit the above-mentioned ophthalmologic services and was also invited to visit the Patronato Nacional de Ciegos under the direction of Dr. B. Courtis, the Santa Lucia Hospital directed by Ibañez Puiggari, the chair of ophthalmology of the Faculty of Medicine of the University of Buenos Aires, directed by Prof. Raul Argañaraz, and the Fernandez Hospital under the direction of Dr. Juan Gallino.

Dr. Alvaro together with Prof. E. B. Demaria visited the eye clinics of the chair of

ophthalmology of the Faculty of Medicine of La Plata, of Rosario, and of Cordoba. In the latter he was received at the local Ophthalmological Society.

In Montevideo, Dr. Alvaro presided at a meeting of the Committee for the Prevention of Blindness of which Prof. Vasquez Barriere is president.

Dr. Juan Verdaguer, Chief of Clinic in the service of Professor Charlin (Santiago de Chile), was appointed Extraordinary Professor of Ophthalmology in the Medical School of the University of Chile. His incorporation thesis dealt with "Detachment of the retina and its treatment."

Dr. M. Uribe Troncoso received an invitation to read a paper at the annual meeting of the Pan-American Medical Association, Washington chapter, and give a lecture on "Gonioscopy, Its development and clinical applications." The meeting was held on March 18, 1944, at the Mexican Embassy, under the presidency of the Ambassador, Dr. F. Castello Nájera, honorary president of the chapter. On the same day Dr. Troncoso was guest of honor at a luncheon at the International Medical Club of Washington, D.C.

Obituary

Dr. Enrique L. Graue, a well-known ophthalmologist in Mexico City, and a former director of the Ophthalmic Hospital of Nuestra Señora de la Luz, died in May, 1943, at the age of 72 years. He was born in Mexico City in 1871. After his graduation, in 1897, he traveled extensively in Europe, attending the London clinics and Paris ophthalmic institutions. He was a fine surgeon and a very charitable man, attending the poor patients in the city and in his hospital with devoted care. He was a member of the Mexican Society of Ophthalmology and Otolaryngology. His oldest son, who has the same name, is also an eye specialist and practices in Mexico City.

NEWS ITEMS

Edited by Dr. Donald J. Lyle
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News items should reach the editor by the twelfth of the month.

Deaths

Dr. Milton R. Barker, Wilmette, Illinois, died February 3, 1944, aged 92 years.

Dr. Edgar Bates, Ogden, Utah, died January 24, 1944, aged 74 years.

Dr. Arthur E. Jessup, Diagonal, Iowa, died January 12, 1944, aged 74 years.

Dr. Waldo N. Lemmon, Hereford, Texas, died January 1, 1944, aged 71 years.

Dr. Frederick M. Spalding, Boston, Massachusetts, died January 24, 1944, aged 73 years.

Dr. Henry R. Slack, LaGrange, Georgia, died January 16, 1944, aged 81 years.

Dr. Roy W. Dunlap, Tulsa, Oklahoma, died January 29, 1944, aged 65 years.

Dr. Halbert H. Acker, Anderson, South Carolina, died January 20, 1944, aged 56 years.

Dr. Horace L. Goodman, Ronceverte, West Virginia, died February 28, 1944, aged 67 years.

Dr. Charles Graef, New York, New York, died February 27, 1944, aged 72 years.

Dr. Arthur C. Haney, Russellville, Arkansas, died December 22, 1943, aged 54 years.

Dr. Frank L. Secoy, Sioux City, Iowa, died January 23, 1944, aged 57 years.

Dr. Daniel H. Bell, Tacoma, Washington, died recently, aged 72 years.

Dr. Joseph W. Ehmer, Crivitz, Wisconsin, died recently, aged 77 years.

Dr. Arthur E. Falkenbury, Whitehall, New York, died January 23, 1944, aged 78 years.

Dr. William J. Holton, Plant City, Florida, died January 7, 1944, aged 60 years.

Dr. Joseph L. Wicks, Evanston, Wyoming, died January 31, 1944, aged 73 years.

Dr. John W. Dean, Glens Falls, New York, died December 18, 1943, aged 75 years.

Dr. Erra D. Stump, Charleston, West Virginia, died March 1, 1944, aged 59 years.

Miscellaneous

The next examination by the American Orthoptic Council will be held in September-October, 1944. The written examinations will be held at various cities in the country on September 7, 1944. Only those passing the written examinations will be permitted to take the oral and practical tests, to be given in Chicago, October 7, 1944. Applications on official forms must be received before August 1, 1944. Address the American Orthoptic Council, 23 East 79th Street, New York 21, New York.

The twelfth semi-annual postgraduate course in "Neuromuscular anomalies of the eyes" will be given at the Children's Memorial Hospital, Chicago, Illinois, by Dr. George P. Guibor, May 7-12, 1944.

As an addition to the growing number of motion pictures in the field of health education a two-reel film, "Eyes for tomorrow," was produced for the National Society for the Prevention of Blindness, 1790 Broadway, New York City. "Eyes for tomorrow" stresses good general health as a prerequisite for good eyesight, besides dealing with the importance of conservation of vision among school children, methods of treatment of glaucoma and trachoma, importance of prenatal care as a means of reducing blindness caused by syphilis and gonorrhea, and the eye hazards of industry. The film, in 16 and 35 mm., will be distributed in the United States by the National Society for the Prevention of Blindness. Prints in 16 mm. are offered for sale at $50.00, or rental at $5.00 per day, exclusive of time in transit.

The American Board of Ophthalmology has announced that examinations will be held in New York on June 2d, 3d, and 5th, and in Chicago on October 5th to 7th. The new address of the Board is 704 Congress Street, Portland, Maine.

The Association for Research in Ophthalmology, Inc., announces its program for the fourteenth scientific meeting, to be held on Tuesday, June 13, 1944, at the Hotel Sherman, Chicago, Illinois.

1. Choline esters with mydriatic and cycloplegic action. Kenneth C. Swan, M.D., and Norman G. White, M.S., University Hospital, Iowa City, Iowa.

2. The dissociation of form and light perception in amblyopia ex anopsia. Hermann M. Burian, M.D., and George Wald, Ph.D., Biological Laboratories of Harvard University.

3. A quantitative test for measuring degree of red-green color deficiency. Louise L. Sloan, Ph.D., AAF School of Aviation Medicine, Randolph Field, Texas.

4. A study of the pathogenicity of diphtheroid bacilli isolated from the human conjunctivae. Charles Weiss, M.D., Isabella H. Perry, M.D., and Marion C. Shevky, A.B., Mt. Zion Hospital, San Francisco, California.

5. Backflow phenomena in aqueous veins of normal and of glaucomatous eyes. K. W. Ascher, M.D., Department of Ophthalmology,

College of Medicine, University of Cincinnati.

6. Effect of chemotherapeutic agents on cell division of the intact and regenerating corneal epithelium following burns and abrasions in the rat. George K. Smelser, M.D., and V. Ozanics, M.D., Columbia University College of Physicians and Surgeons, New York.

7. Evaluation of the use of penicillin in military ophthalmology. John G. Bellows, Major (M.C.), Billings General Hospital, Fort Benjamin Harrison, Indiana.

Frederick C. Cordes, Chairman
Brittain F. Payne, Major (M.C.)
Secretary (Program)
Conrad Berens
Acting-Secretary-Treasurer

Societies

The annual congress of the Ophthalmological Society of Egypt was held at the Memorial Ophthalmic Laboratory, Giza, Egypt, on March 15th and 16th. The program consisted of a symposium on "Ulcers of the cornea."

The Florida Medical Association held its seventy-first annual meeting at Saint Petersburg on April 13th and 14th. Among the speakers was Dr. Walter I. Lillie, Philadelphia, who lectured on "Fundus changes in arterial hypertension." Included in the specialty groups which met during the session was the Florida Society of Ophthalmology and Otolaryngology.

At the annual session of the South Carolina Medical Association Dr. Roderick MacDonald, Rock Hill, presented a paper on "Headache from an eye, ear, nose, and throat standpoint."

The Brooklyn Ophthalmological Society held its regular meeting on April 20th. The scientific program consisted of the following papers: "The pathology of congenital hydrophthalmos and allied conditions" by Drs. Bernard Samuels and Edward Burchell, and "Surgical treatment of congenital glaucoma," by Dr. John H. Dunnington.

Personals

The Warren Triennial Prize of $500, presented by the Massachusetts General Hospital, was awarded to Dr. David G. Cogan, Dr. V. Everett Kinsey, and Mr. Erwin O. Hirsch, of the Howe Laboratory of Ophthalmology, Harvard Medical School, for the essay "Physiological studies on the cornea."

The University of Minnesota Medical School, Minneapolis, has appointed Dr. Erling W. Hansen, former clinical assistant professor of ophthalmology, to the position of clinical professor and director of the division of ophthalmology at the university.

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